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A.M.A. ARCHIVES OF

OTOLARYNGOLOGY

Blood Loss During Tonsillectomy
Hueston C. King and Stratton R. Story

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Manipulation of the Labyrinth Windows and Their Surroundings in Tympanoplasty
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Vital Staining of Cervical Lymphatic Nodes by Means of Direct Sky Blue
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Primary Adenoidcystic Carcinoma of the Esophagus
Raul A. Marcial Rojas and Luis A. Vallecillo

Five-Year Analysis of External Ethmoid and Frontal Sinus Operations
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Benign Hypertrophy of Masseter Muscle
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The Responsibility of the Speech Therapist to the Laryngectomized Patient
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The Effect of Phenylephrine (Neo-Synephrine) on Pure-Frequency Audiometric Scores
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Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Internal Ear (To Be Concluded)
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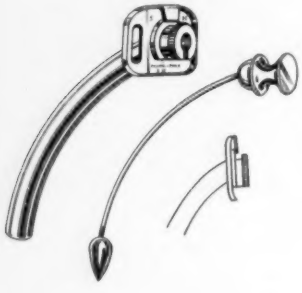

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TABLE OF CONTENTS

VOLUME 70 AUGUST 1959 NUMBER 2

ORIGINAL ARTICLES

Blood Loss During Tonsillectomy
Capt. Hueston C. King (MC) and Capt. Stratton R. Story (MC), U. S. Army 153

The Clinical Significance of a Lump in the Throat
G. Edward Tremble, M.D., Montreal 157

The Effect of Cigarette Smoke on Ciliary Activity in the Upper Resiratory Tract
Tore Dalhamn, Tomteboda, Sweden 166

Effect of Conventional Fenestration on Ménière's Disease
Yrjö Meurman, M.D., Helsinki, Finland 169

Manipulation of the Labyrinth Windows and Their Surroundings in Tympanoplasty
Shuji Goto, M.D., Nagoya, Japan 185

Vital Staining of Cervical Lymphatic Nodes by Means of Direct Sky Blue
Ryuichi Kubo, M.D., Kagoshima, Japan 192

Primary Adenoidcystic Carcinoma of the Esophagus
Raul A. Marcial Rojas, M.D., and Luis A. Vallecillo, M.D., San Juan, P. R. 197

Five-Year Analysis of External Ethmoid and Frontal Sinus Operations
Charles R. Smathers, M.D., New York 202

Benign Hypertrophy of Masseter Muscle
H. Oppenheim, M.D., and Wing, M.D., Louisville 207

The Responsibility of the Speech Therapist to the Laryngectomized Patient
Maurice H. Miller, Ph.D., Brooklyn 211

AUDIOLOGY SECTION

The Effect of Phenylephrine (Neo-Synephrine) on Pure-Frequency Audiometric Scores
E. Thayer Curry, Ph.D.; Floyd Boys, M.D., and Donald B. Stone, M.S., Urbana, Ill. 217

PROGRESS IN OTOLARYNGOLOGY

Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Internal Ear (To Be Concluded)
Bruce Proctor, M.D., Detroit, with the Collaboration of Michel Portmann, M.D., Bordeaux, France; Enrico Bozzi, M.D., Milano, Italy; Jerzy Szpunar, M.D., Krakow, Poland, and Thea A. Hennecke, M.D., Windsor, Ont., Canada 220

SOCIETY TRANSACTIONS

Chicago Laryngological and Otological Society 271

REGULAR DEPARTMENTS

Abstracts from Current Literature 277

Books:
Book Received 284
Book Reviews 284

Directory of Otolaryngological Societies 285

A. M. A.
Archives of Otolaryngology

VOLUME 70 AUGUST 1959 NUMBER 2

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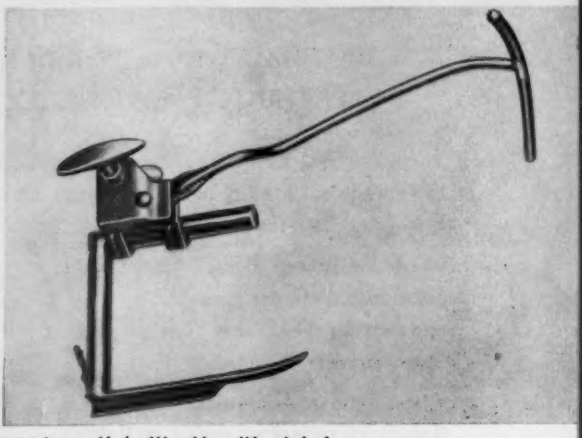

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A.M.A. ARCHIVES OF
OTOLARYNGOLOGY

Blood Loss During Tonsillectomy

CAPT. HUESTON C. KING (MC) and CAPT. STRATTON R. STORY (MC), U. S. Army

For over 2,000 years tonsillectomy has been the center of heated disputes involving both laymen and physicians.[1] The procedure has been praised, damned, disparaged, and advised, but, whether for or against or merely intrigued by some ramification of the problem, few of those interested have remained neutral or silent. Boies,[2] in reviewing the literature on tonsillectomy and the problems related to it, found over 100 papers a year recorded in the "Quarterly Cumulative Index Medicus" between 1942 and 1946, despite the marked decrease in medical writing brought about by the war.

The question of whether or not to perform the tonsillectomy and adenoidectomy resolves itself into two opposing parts. On the one hand is the benefit to be expected from removal of the diseased tonsils and adenoids; on the other, the inherent risk involved. Indications for tonsillectomy and adenoidectomy have been discussed extensively by a wide range of writers,[3-5] and most authorities are essentially in agreement. The dangers in removing the tonsils and adenoids are equally well known, with hemorrhage leading the field by a wide margin.

Submitted for publication Nov. 21, 1958.

Otolaryngology Service, Department of Surgery, Walter Reed Army Hospital, Walter Reed Army Medical Center, Washington 12, D. C. Present address of Capt. King: United States Army Hospital, United States Military Academy, West Point, N. Y.

With the ever-present threat of severe bleeding, it would be helpful to know how much blood is lost in a clinically uncomplicated tonsillectomy and adenoidectomy. This would provide a base line and give at least an idea of how much reserve can be expected. The surgeon would also like to know whether measurements of hemoglobin and hematocrit provide an accurate means of estimating the amount of blood loss. With this in mind, a study was made at Walter Reed Army Hospital of 66 children undergoing tonsillectomy and adenoidectomy with general anesthesia.

Method of Study

The 66 children were unselected cases undergoing surgery between September, 1957, and June, 1958. One week before the operation a hemoglobin and hematocrit determination was made on each child, as a part of the regular work-up. All surgery was performed in either the Rose or the supine position, and general anesthesia was used. The adenoids were removed with the Shambaugh adenotome and Barnhill curets. The peritonsillar mucosa was then incised with a No. 12 Bard-Parker blade, and the tonsil dissected free from above downward with the Hurd elevator and Fisher knife, and finally removed with a Tyding snare. Hemostasis was secured in the manner described by Fox,[6] with plain absorbable (gut) surgical suture ties and pressure. To measure the blood lost during surgery, 500 cc. of isotonic saline was carefully measured and placed in a sterile basin on the Mayo stand beside the operating table. This was used for flushing the suction tubing during and at the conclusion of the operation. Only blood was aspirated through this suction system; cases were discarded in which vomitus or

Data on Sixty-Six Cases

Case	Preoperative		Postoperative				Age	Wt., Lb.	Blood Loss, Cc.
	Hgb.	H'crit.	Hgb.	Change	H'crit.	Change			
1	13.0	41.0	12.3	−0.7	33.5	−7.5	8	74.00	232.4
2	12.9	37.0	10.4	−2.5	33.0	−4.0	8	44.50	226.4
3	12.5	38.0	11.5	−1.0	33.0	−5.0	7	36.75	217.8
4	13.1	38.0	11.5	−1.6	35.0	−3.0	6	51.50	194.6
5	12.9	38.0	12.1	−0.8	34.0	−4.0	5	45.00	180.6
6	13.4	38.0	12.4	−1.0	34.0	−4.0	13	117.00	167.0
7	13.6	38.5	13.0	−0.6	37.0	−1.5	6	51.50	138.1
8	12.5	36.0	11.4	−1.1	32.0	−4.0	6	41.50	138.0
9	10.7	34.0	11.8	+1.1	33.0	−1.0	7	49.50	137.4
10	14.8	38.0	13.9	−0.9	38.0	0.0	7	42.50	127.0
11	10.9	34.0	12.3	+1.4	32.5	−1.5	9	54.00	126.4
12	13.3	39.0	11.5	−1.8	36.0	−3.0	5	48.00	118.4
13	14.6	43.5	12.9	−1.7	38.0	−5.5	11	137.00	116.2
14	13.4	34.0	12.1	−1.3	36.5	+2.5	7	46.00	115.4
15	14.2	38.5	13.9	−0.3	40.0	+1.5	6	42.00	113.6
16	12.2	39.0	12.4	+0.2	35.0	−4.0	5.5	53.00	113.0
17	12.2	38.0	12.0	−0.2	35.0	−0.3	4	41.00	111.8
18	13.6	41.0	12.3	−1.3	36.0	−5.0	6	53.50	103.6
19	12.9	40.0	12.3	−0.6	35.0	−5.0	5	40.75	99.6
20	12.9	39.0	12.4	−0.5	37.0	−2.0	10	80.00	98.6
21	11.3	32.0	12.5	+1.2	37.5	+5.5	7	60.00	95.0
22	13.4	38.0	12.4	−1.0	36.0	−2.0	8	57.00	85.8
23	14.6	39.0	12.3	−2.3	36.0	−3.0	8	57.75	84.2
24	13.4	41.0	13.3	−0.1	42.0	+1.0	10.5	83.25	84.0
25	11.5	36.0	12.3	+0.8	34.0	−2.0	11	71.50	80.8
26	15.0	42.0	11.0	−4.0	33.5	−8.5	7	57.00	80.6
27	13.6	40.0	13.1	−0.5	37.0	−3.0	8	64.00	79.4
28	11.7	38.0	13.0	−1.3	40.0	+2.0	4.5	58.00	79.0
29	12.5	37.0	12.8	+0.3	38.0	+1.0	7	47.50	78.8
30	11.9	39.0	11.6	−0.3	36.0	−3.0	8	59.00	78.6
31	11.2	31.5	11.5	+0.3	32.0	+0.5	4.5	37.00	73.2
32	11.2	37.5	11.1	−0.1	34.0	−3.5	5	43.00	73.2
33	11.2	35.0	13.1	+1.9	40.0	+5.0	6	50.00	73.2
34	13.7	42.0	13.1	−0.6	37.0	−5.0	8	79.00	71.8
35	13.6	39.5	12.4	−1.2	36.0	−3.5	11	131.50	70.6
36	11.8	31.0	13.1	+1.3	37.0	+6.0	6	68.00	68.6
37	13.3	43.5	13.8	+0.5	42.5	−1.0	11	80.50	68.0
38	12.9	39.0	12.1	−0.8	37.0	−2.0	7	66.75	67.0
39	14.4	40.5	11.9	−2.5	37.0	−3.5	7	43.00	66.6
40	13.6	39.0	12.1	−1.5	34.0	−5.0	6	37.00	61.6
41	12.4	35.0	11.5	−0.9	35.0	0.0	7	59.50	61.2
42	11.7	37.5	12.1	+0.4	34.5	−3.0	5	43.50	59.2
43	12.5	36.5	13.3	+0.8	39.0	+2.5	5.5	43.25	58.0
44	10.7	39.0	12.1	+1.4	37.0	−2.0	7	54.00	54.8
45	11.4	40.0	11.9	+0.5	33.5	−6.5	5	45.00	54.6
46	14.2	37.5	11.5	−2.7	35.0	−2.5	8	68.75	54.0
47	12.2	38.5	12.8	+0.6	38.0	−0.5	6	58.00	51.2
48	11.9	35.0	11.0	−0.9	37.0	+2.0	6.5	52.50	48.6
49	11.1	36.0	10.1	−1.0	30.0	−6.0	5	45.00	43.0
50	14.8	40.0	13.7	−1.1	39.5	−0.5	5.5	48.25	39.8
51	12.9	41.0	12.8	−0.1	37.0	−4.0	6	50.50	38.8
52	11.9	35.0	12.3	+0.4	36.0	+1.0	6	46.00	38.4
53	12.0	37.5	12.6	+0.6	37.0	−0.5	6	37.00	38.4
54	12.7	40.0	12.6	+0.1	38.0	−2.0	6	51.00	38.0
55	14.4	44.0	11.6	−2.8	39.0	−5.0	5	43.00	37.6
56	13.8	41.0	12.6	−1.2	39.0	−2.0	7	49.00	37.4
57	13.1	37.0	12.6	−0.5	35.0	−2.0	5	39.25	36.8
58	13.3	40.0	12.8	−0.5	40.0	0.0	6	48.50	34.0
59	12.9	39.0	11.0	−1.9	34.0	−5.0	4	38.75	33.8
60	11.7	40.0	13.0	+1.3	36.0	−4.0	4	39.00	32.2
61	14.6	40.0	11.6	−3.0	35.0	−5.0	6	41.25	32.2
62	12.2	35.0	11.1	−1.1	36.0	+1.0	8	57.50	29.8
63	13.4	41.0	13.1	−0.3	41.0	0.0	7	63.75	26.8
64	12.2	38.0	11.8	−0.4	37.0	−1.0	5	51.50	26.2
65	14.6	43.0	14.7	+0.1	46.0	+3.0	9	61.25	25.0
66	12.9	37.0	11.9	−1.0	36.0	−1.0	6	46.00	21.8

appreciable quantities of other secretions were inadvertently aspirated. After surgery the content of the suction bottle was measured. Experimental aspiration of a measured quantity of water into the suction bottle showed no measurable amount of fluid remaining in the suction system, and so at surgery all fluid aspirated in excess of 500 cc. was considered to be blood.

The sponges used during surgery were soaked in 200 cc. of distilled water to produce the greatest possible degree of hemolysis and were then squeezed dry. The soak fluid was sent to the laboratory for hemoglobin determination, with the determination being made as though the fluid were whole blood. The figure obtained was converted to cubic centimeters of the patient's blood by the following computation:

1. Hemoglobin value from laboratory is actually grams of hemoglobin per 100 cc. of fluid. Therefore, twice this figure equals the total hemoglobin in 200 cc., or the total amount of hemoglobin on the sponges.

2. As the colorimeter used in hemoglobin determination measures the amount of hemoglobin per 100 cc. of whole blood,

$$\frac{\text{total hgb. on sponges}}{\text{blood (cc.) on sponges}} = \frac{\text{patient's preoperative hgb.}}{100 \text{ cc.}},$$

or,

$$\frac{\text{total hgb. on sponges} \times 100}{\text{patient's preoperative hgb.}} = \text{blood (cc.) on sponges.}$$

The total blood loss for the operation was then computed by adding the amount of blood aspirated through the suction tubing to that released from the sponges.

As a part of the study, hemoglobin and hematocrit determinations were made again 24 hours after surgery. Children who had bled during these first 24 hours, as well as those who had run a persistent fever, were dropped from the records, leaving for study only uncomplicated tonsillectomies and adenoidectomies. Each child's weight and age were recorded before surgery to determine whether a relationship existed between either factor and the amount of blood lost at surgery.

Comment

The table shows all 66 cases, arranged in order of blood loss, with hemoglobin and hematocrit and their change following surgery. The age and weight of each child are also recorded. While a general tendency is seen for both hemoglobin and hematocrit to fall slightly after surgery, this fall was not consistent and in nine of the cases both figures rose slightly. In 17 more cases, one figure fell while the other rose. This represents approximately one-fourth of the total number of cases. If all cases not showing a symmetrical fall in both hemoglobin and hematocrit are considered, including four cases in which the hematocrit remained unchanged while the hemoglobin fell slightly, the figure totals over 45% of the series. This large group is distributed through the series in a random fashion and follows no predictable pattern. It is readily seen that, while hemoglobin and hematocrit determinations may be indicated in evaluating hemorrhage, such tests taken the morning after surgery, which is the customary time of discharge from the hospital, are not to be considered a reliable means of estimating the amount of blood lost.

Figure 1 shows the actual blood loss in graphic form. The average loss was 82.5 cc., with most cases grouped between 20 and 140 cc. This average is about midway between the 51 cc. average reported by James T. King,[7] in 1954, and the 128 cc. average reported by McKenty,[8] in 1937. As in their series, a fairly wide range between the extremes of loss appeared in the group

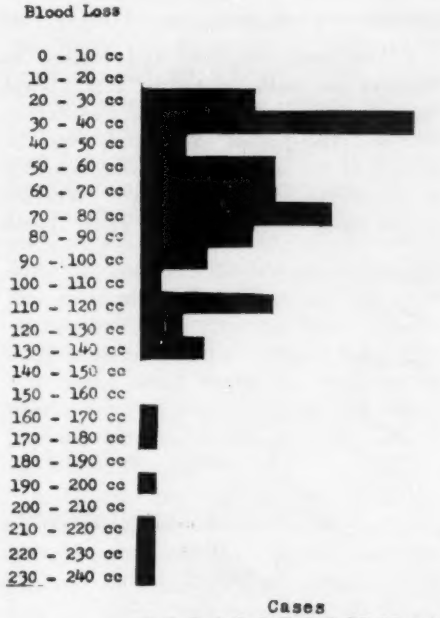

Figure 1

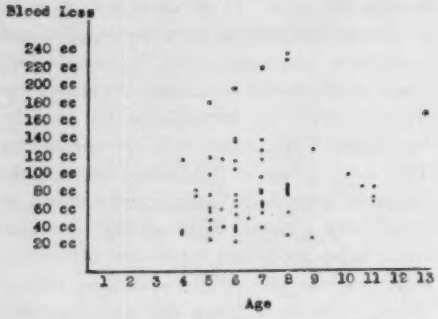

Figure 2

studied. Neither of the above investigators reported studies of the hemoglobin or hematocrit, making further comparison impossible.

Figures 2 and 3 are scattergrams, plotting blood loss against the age and weight of the patient, respectively. The children's ages ranged from 4 to 13 years, with 79% being between 5 and 8 years of age. Their weights ranged from 39 to 137 lb. Little tendency is seen for the figures plotted to follow any specific pattern.

Conclusions

1. We were surprised and pleased to discover the small amount of actual blood loss. In only one case did the loss exceed 200 cc. The patient, of course, must be treated as the individual situation demands if postoperative bleeding occurs. Whether or not blood transfusions are given should depend on the clinical condition of the particular patient.

2. Although at this hospital and others preoperative and postoperative determinations of hemoglobin and hematocrit are routinely made for the evaluation of uncomplicated cases, this series indicates that such determinations do not truly reflect the amount of blood loss. In a significant number of cases, the postoperative hemoglobin and/or hematocrit was found to be higher than the preoperative value. In part, at least, dehydration was felt to be responsible for this apparent rise. It could be assumed, however, that a marked fall in either hemoglobin or hematocrit, or both, would be significant and that the patient should be evaluated for hidden bleeding.

3. An attempt was made to correlate the amount of blood loss with either the age or the weight of the patient, but no relationship could be demonstrated.

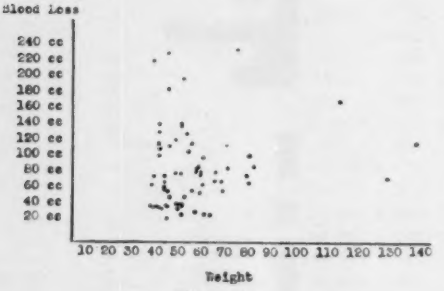

Figure 3

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The Clinical Significance of a Lump in the Throat

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At the present time there is a tendency on the part of laryngologists in general to consider any complaint of a lump in the throat by a patient to be functional in origin. The very term globus hystericus itself suggests an anxiety state. According to Weiss and English,[1] "The hysterical symptom of swallowing over a lump is one of the most widely recognized indications of nervous illness." Jacobson [2] demonstrated with x-ray studies that the sensation of a lump is produced by an esophageal spasm. This spasm, which is the motor expression of an underlying emotional disturbance, may also cause gagging and choking.

Since the war, unsettled world conditions are undoubtedly reflected on individual persons, and more patients seek advice for indefinite aches and pains than ever before. The growing tensions of every day living are often shown, in our field, by abnormal sensations in the pharynx, such as burning, itching, dryness, hypersalivation, or foreign body. For instance, Furstenberg and Crosby [3] made the statement that a large percentage of patients who consult an otolaryngologist with the complaint of dry mouth suffer from some form of depression. As another example, Lederer,[4] in listing the causes of hypersalivation, mentioned "nervous instability" and "severe emotional disturbances."

The sensation of a lump in the throat is such a common complaint, especially among women, that we are likely to treat it lightly. However, it is dangerous to assume that this symptom is purely functional without a careful examination with every means at our disposal. Local inflammatory and neoplastic processes about the pharynx and tongue not infrequently lead to pharyngeal and obstructive spasms. These symptoms are, however, neurological in nature, associated with definite organic lesions.

Acute inflammatory infections are not as a rule difficult to observe and diagnose, but sometimes an early malignancy at the base of the tongue remains "silent" for weeks or months. A case like this presented itself a year after being examined by two reputable physicians. The main complaint in the early stages was the feeling of a lump in the throat. A routine nose and throat examination was negative, as was a general physical examination, including a barium swallow, a chest x-ray, a bronchoscopy, and an esophagoscopy. In the absence of any positive findings, it was assumed that the "lump" was functional in origin. As the symptoms did not subside, it was thought that a chronic low-grade lingual tonsillitis was responsible for the pain and dysphagia that developed. Some time later a node appeared behind the left sternomastoid muscle, but unfortunately the patient was in a distant country, and this was neglected. Eventually when the node broke down and suppurated and one was palpated behind the right sternomastoid, he returned home. At this stage a sloughing crater larger than a 10-cent piece could easily be seen, in the mirror, on the base of the tongue at the level of the tip of the epiglottis just to the left of the midline.

A biopsy specimen showed squamous-cell carcinoma. This case is cited to bring out one or two helpful points and not with the idea of being critical. Anyone might have overlooked this growth at the beginning, when the only complaint was a lump

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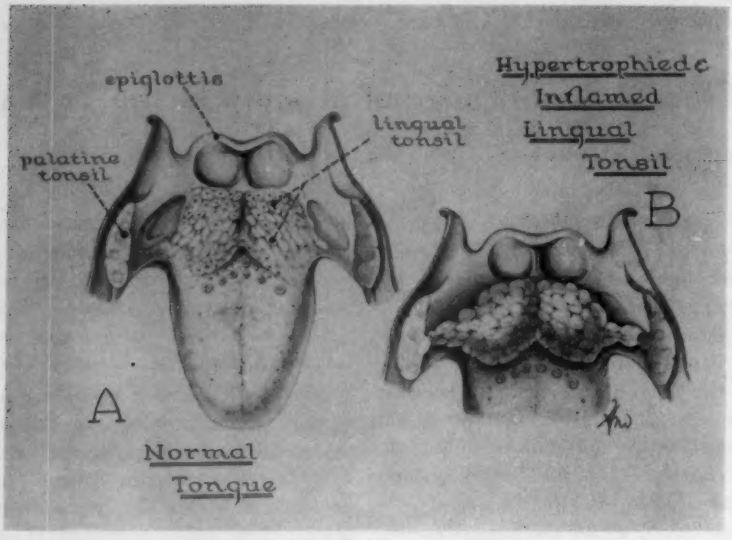

Figure 1

in the throat. However, it should always be kept in mind that an early carcinoma at the base of the tongue can usually be palpated with the gloved finger long before any ulceration is seen in the mirror. Unless such a malignancy is recognized early and radical treatment carried out, the condition is hopeless. Unfortunately, the case described happened to be a personal friend, which made it all the more upsetting. In the above-mentioned case, lingual tonsillitis was considered for a time to be the cause of the patient's symptoms.

The usual complaints of an inflammation of the lingual tonsils are pain and discomfort on swallowing. The severity of the symptoms depends on whether the condition is acute or chronic. Lymphoid follicles at the base of the tongue on each side of the midline are easily visualized in the mirror. It should not be difficult for a laryngologist to determine whether they are within normal limits or hypertrophied. Unfortunately, nose and throat surgeons at times tend to neglect this area.

The symptoms vary considerably when the lingual tonsils are inflamed or enlarged and have been enumerated elsewhere. To quote from a recent article:

> A constant feeling of wanting to clear the throat or to swallow are common complaints. Pain low down in the throat at the level of the hyoid bone is very suggestive of this condition. At times, patients say there is a fullness or tightness of the throat as though they were swallowing over a lump, symptoms often associated with globus hystericus. When these complaints occur in middle aged women they are often thought to be due to the menopause.
>
> In lingual tonsillitis:
>
> Discomfort on swallowing is usually just above the thyroid cartilage and to one or other side of the midline, depending on whether one or both sides are involved. When the palatine tonsils are affected, the sore throat is higher and the cervical glands below the angle of the jaw are enlarged and tender.[6]

Lingual tonsillitis, instead of being a self-limited infection of three or four days duration, is likely to persist and become subacute. At times a low-grade infection can become chronic and go on for weeks or months. Occasionally the masses of lymphoid tissue press on the epiglottis and interfere with swallowing. Sometimes voice changes also occur.

When the lingual tonsils become inflamed and swollen repeatedly, the sinuses should be suspected and carefully examined. Also, as with any persistent sore throat, infectious

mononucleosis should always be kept in mind.

The treatment of hypertrophied lymphoid nodules consists in reducing the size of the heaped-up tissue. This can be accomplished with electric cautery, but a disadvantage is the charring effect that results, which often causes dryness and crusting. Electrocoagulation under local anesthesia is much superior. Occasionally, if the lymphoid masses are tremendously enlarged, it is more satisfactory to remove them by dissection and snare. The bleeding is not as a rule severe and can usually be controlled by pressure. Unfortunately, hypertrophied tissue in this region has not been fully appreciated by laryngologists in general.

Varicosities are frequently associated with enlarged lingual tonsils and are occasionally responsible for a persistent cough. If a nervous patient is aware of their presence, he is likely to develop a neurosis which gives rise to a feeling of a foreign body, sometimes with increased salivation. The veins at the base of the tongue are usually very superficial. When hypertrophied veins and varicosities are present, they appear as prominent, tortuous vessels, which can easily be seen in the laryngeal mirror. Their cause is unknown, unless chronic irritation, such as repeated attacks of pharyngitis, play a part. There has never been any definite proof that varices at the base of the tongue are associated with varicose veins elsewhere, but some laryngologists feel that they are part of certain systemic infections.

Unless there is something definite, such as venous oozing on exertion or coughing, varicosities at the base of the tongue should be left strictly alone. If troublesome bleeding occurs from time to time, the offending varices can be destroyed by electrocoagulation under surface anesthesia with cocaine or tetracaine (Pontocaine). Although lingual varices rarely cause the sensation of a lump in the throat, they should always be kept in mind.

Many years ago Watson-Williams [6] described paresthesia as a perverted sensation, giving rise to a feeling of a foreign body in the pharynx.

In the absence of any organic lesion, he divided these "lumps" into three fairly distinct groups. They have been referred to recently again by Mills.[7]

(1) *The lump which moves up and down.*
This is a common complaint after an influenzal attack and is experienced by both men and women. The inflamed membranes of the nose and throat are part of the general infection and this often takes a week or two to subside. During this time the patient is sometimes conscious of a fullness in the throat. This is due to the up and down movement of the posterior surface of the cricoid against the back of the inflamed pharynx during swallowing. As the inflammation lessens the feeling of the lump disappears.

(2) *The lump with aching.*
This is most often found in women and occurs at all ages. The mucosa of the nose and throat is anaemic looking and resembles that seen in atrophic rhinitis. Owing to the pale, thin, membrane and the small turbinates, the nasal fossae are more widely open than usual. As a result it is often possible to see the nasopharynx through the anterior nares. Again due to the thinness of the membranes, the vessels in the submucosa of the pharynx are very prominent and easily seen. This combination of symptoms is often associated with thyroid insufficiency. A metabolism test is indicated in all these cases. At times, however, even in the presence of a normal metabolic rate small doses of thyroid extract twice a day are often helpful. General systemic treatment, including iron and vitamins is also indicated.

(3) *The emotional lump.*
The typical case is the so-called globus hystericus and occurs as mentioned previously in those whose nervous systems are not too stable. Although predominantly found in women, it is often seen in men. The hard working, middle-aged executive not infrequently consults the laryngologist fearing a malignancy of the throat, perhaps after hearing of a friend with a similar condition. This might well be expected considering the publicity given cancer campaigns in the press, on the radio and on television, stressing the importance of an early diagnosis. Whether or not the patient admits it, there is always the suspicion on the part of the laryngologist of some underlying factor or emotional strain as the basis of his complaint.

Excessive smoking and drinking cause a law-grade chronic inflammation of the respiratory tract and aggravate the condition. Fixed-tissue changes occur, with a permanent dilatation of the blood vessels

of the nasal and pharyngeal mucosa, and as a result the mucus becomes thick and sticky. The overburdened cilia have difficulty functioning under these conditions, and the constant clearing of the throat adds to the patient's discomfort. Under such conditions it is not unusual for certain patients to become apprehensive at first and later obscessed with the idea of a growth. Most laryngologists have a number of patients who report of their own volition once or twice a year to be assured that there is no sign of cancer of the throat. In view of this it is not difficult to realize why the manufacture and sale of tranquilizers has increased to such an extent, even in the practice of otolaryngology.

Apart from any functional disorder a number of systemic diseases are often accompanied by perverted sensations in the throat, such as burning, itching, tightness, rawness, or a feeling of a foreign body. Some of the common ones listed by Thomson and Negus [8] are anemia, tuberculosis, gout, dyspepsia, and, in hypochondriacs or those who use tobacco or alcohol to excess, conditions which produce local congestion.

A few remarks are necessary about chronic pharyngitis, as the end-results of this condition so often produce the sensation of a lump in the throat. As a rule, chronic infections of the pharynx follow repeated attacks of acute pharyngitis, which is a common complaint in the winter months. Constant irritation, such as occurs in sinusitis and in some occupations, such as the fur trade, cloth cutting, and stone masonary, often predisposes to chronic pharyngitis. Irritating fumes, chemical agents, and dust, as well as the abuse of tobacco and alcohol, aggravate the condition. Allergy must always be kept in mind as a possible cause.

Perhaps the most frequent complaint of the patient is that he feels there is something in his throat that is bothering him. The constant clearing keeps it irritated, dry, and inflamed and sets up a vicious circle. The rasping and scraping increase the redness, and the redness causes the patient to make repeated ineffectual attempts to clear the throat. Microscopically this inflammation shows a gradual increase in the connective tissue cells of the submucosa, with a resultant hyperplasia of these cells which persists. All the mucles of the pharynx become hypertrophied, which often gives the patient a sense of fullness in the throat. The follicles and all the lymphoid tissue in the submucous layer are increased. The irregular patchy areas on the posterior pharyngeal wall are made up of masses of lymphoid cells which become elevated and prominent. As there is a tendency for the lymphoid cells to be concentrated around the openings of the mucus-secreting glands, shrinkage occurs, and the ducts become obstructed with lessening of secretion. The glazed or varnished appearance of the pharynx is the result of the lack of mucus. Owing to the absence of cilia, thick, gummy exudate collects on the posterior wall, and the patient is forced to get rid of it by hawking and coughing.

This granular type with irregular heaped-up discrete masses is seen oftenest in young adults, while the hypertrophic form, including the lateral bands, is much more frequent in the third and fourth decades.

A number of years ago Turner [9] mentioned granulations on the posterior pharyngeal wall as giving rise to a feeling of a "lump" in the throat. More recently Lederer [10] suggested that in many of the so-called cases of "globus" a local lesion could be found in the throat to account for the symptoms. He cited inflamed lymphoid follicles as the commonest cause, although any lesion behind the posterior pillar or in any area where two folds of mucous membrane rub together during swallowing can also be responsible. This, of course, includes the lateral band on each side which when present extends from the posterior lip of the opening of the Eustachian tube down the side of the pharynx just behind the posterior pillar.

According to Thomson and Negus,[11] "The bands seen in lateral pharyngitis are

by some considered to be similar lymphoid collections, by others they are regarded as chiefly formed by hyperplasia of the submucous connective tissue, while some attribute them to muscular hypertrophy in the salpingopharyngeal folds."

The posterior pharyngeal wall is composed of a surface layer of stratified, squamous epithelium on top of a fibrous coat. These relatively thin coats lie on a muscular layer. The lymphoid tissue, which is identical to that found elsewhere, lies in the fibrous or connective tissue lining. When these solitary follicles become raised and prominent, they are considered abnormal.

In his book, "The Pharynx," Hollender [12] stated:

> The lymphoid tissue of the lateral folds is found in the fibrous mucosa and does not differ from that of the posterior wall. Instead of isolated nodules, a column or fold is seen on each side of the pharynx. On account of its position along the lateral wall, the fold resembles a reduplication of the posterior pillar. When the nodules of the lateral folds are visibly enlarged, they are to be regarded as pathologic. Marked hypertrophy of the bands, which sometimes follows removal of the faucial tonsils and adenoids, may be purely compensatory and harmless, though some clinicians will take issue with this view. However, when subacute or chronic infection produces hypertrophy accompanied by symptoms, permanent reduction of the masses is indicated.

The reason for citing the views of various authorities is to stress the fact that too often the posterior pharyngeal wall and lateral bands are neglected. If this area were carefully inspected routinely the cause of many abnormal sensations in the throat would be found.

When a patient reports, complaining of a lump in the throat, the average otolaryngologist after a careful history examines the ears, nose, and pharynx in a routine manner. He looks over the tonsils to see whether they are inflamed or scarred and then inspects the nasopharynx with the postnasal mirror and perhaps the nasopharyngoscope. After examining the valleculae and epiglottis he centers his attention on the cords and arytenoids. Unless some lesion is found to explain the discomfort, he is likely to think that the underlying cause is due to some emotional upset. Whether or not he suggests a barium swallow or a metabolism test depends on the individual physician.

Acute follicular pharyngitis is not difficult to diagnose, as there are usually localizing symptoms. The swollen and inflamed lymphoid follicles scattered over the posterior wall are very prominent and easily seen. When the lymphoid tissue in the salpingopharyngeal folds is enlarged and congested the lateral bands are ropey in appearance. Although this is easily visualized by the examiner, he is often surprised on questioning to learn that the patient does not complain of the back of his throat. This has been ably described by Thomson and Negus. (In writing an article like this it is almost impossible not to refer to them frequently.)

On the subject of the patient's complaints not necessarily going hand in hand with the appearance of the pharynx, they stated: "There is no necessary proportion between the subjective symptoms of pharyngitis and the pathological changes found. The converse of this statement is equally true, for many individuals may present all the physical signs of pharyngitis and yet be entirely free from discomfort or disturbance." [9]

In a typical history many of these patients complain of a fullness or tightness in the throat, a feeling at times as though they were swallowing over a lump. On the other hand there is no difficulty at meal times; in fact, food often relieves the sensation.

It is needless to say that when a patient reports to a laryngologist with these complaints a thorough examination should be carried out. During the routine check-up the lymphoid follicles on the pharyngeal wall should be inspected as well as the lateral bands. If they are inflamed or enlarged it would seem wise to probe them gently with a moist cotton-tipped applicator. This often gives a reflex feeling low down in the throat simulating the patient's complaints. These trigger areas, although de-

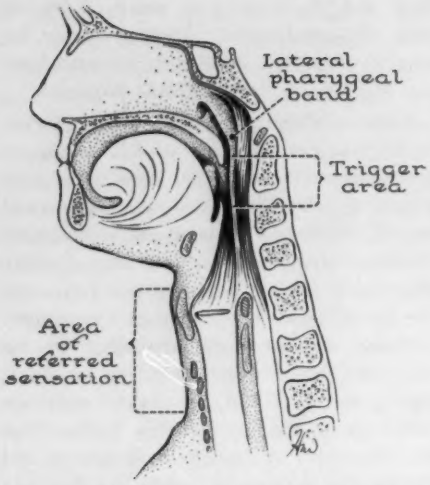

Fig. 2.—Side-view, showing lateral pharyngeal band. When this trigger area is inflamed and swollen, stimulation often produces the same sensation as that complained of by the patient.

scribed years ago are not as well known as they should be to otolaryngologists. In the absence of any other obvious lesion these spots should be tested because, if the surgeon is unable to find a definite explanation for his discomfort, the patient is likely to lose faith in his doctor and become apprehensive.

If one suspects inflamed or hypertrophied follicles on the posterior wall, gentle probing of the nodules often elicits the sensation the patient complains of, and he points to the region of the cricoid cartilage.

When the lateral pharyngeal band is responsible, slight pressure along the muscle on that side is frequently referred to a spot low down in the throat near the anterior border of the sternomastoid muscle. In cases where both lateral bands are affected, stimulation of these areas causes an abnormal feeling in the center of the neck over the cricoid and trachea.

In other words, when sensitive nodules over the back of the pharynx or both lateral bands are stimulated, often an abnormal feeling is elicited in the region of the cricoid cartilage.

When one side alone is involved the sensation is unilateral, low down on the affected side. If the lateral pharyngitis extends upward to the Eustachian tube orifice, ear symptoms are commonly part of the syndrome. The trigger zone is just behind the edge of the soft palate at the tubal opening. (It is a well-known fact that one of the earliest symptoms of a carcinoma of the nasopharynx in the neighborhood of the tube is a fullness or pain in the ear on that side.) If these areas are kept in mind the source of many indefinite aches and pains in the front and sides of the neck can be ascertained. The symptoms [13] of "lateral pharyngeal band syndrome" have been listed by Pastore as follows:

(1) The presence or absence of a history of an upper respiratory tract infection,

(2) Discomfort in the center or side of the pharynx associated with an abnormal feeling, such as itchiness, scratchiness, or lumpy sensation,

(3) A demonstrable trigger zone, generally along the lateral band and corresponding to the referred site of discomfort.

If the symptoms are relieved by the application of general and local treatment the diagnosis is confirmed.

In view of the fact that the majority of patients with granular pharyngitis, espe-

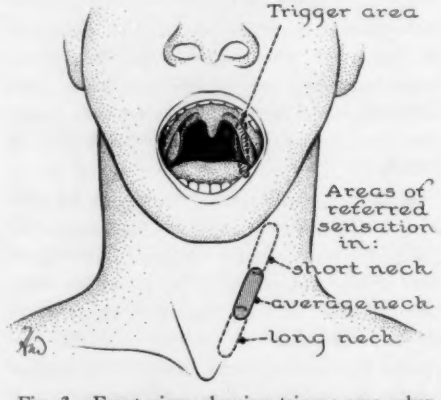

Fig. 3.—Front view, showing trigger area when hypertrophied lateral pharyngeal band is responsible for patient's symptoms. Probing the lateral band frequently gives the same abnormal symptoms along the border of the sternomastoid muscle. When both sides are involved the patient tends to localize the referred sensation to the midline.

cially where the lateral bands are involved, show a nervous instability, local treatment should only be carried out as a last resort. It is far better to have the patient checked thoroughly, and, if everything is negative, he can be assured that there is no sign of malignancy. This is often all that is required. Adequate doses of iodine, iron, and vitamins are sometimes helpful. Unfortunately, there is a tendency on the part of eager laryngologists to apply strong silver nitrate, trichloracetic acid, phenol, x-ray, galvanic cautery, or electrocoagulation to reduce the redundant tissue, but it is wiser not to center the patient's attention on those areas unless all other methods fail.

When asked his opinion on this subject, Dr. Francis Lederer [14] expressed his feelings a little differently:

Undoubtedly the people who are aware of the presence of enlarged lateral bands and an abundance of lymphoid follicles in the mesopharynx have such feelings, or sensations as to cause a "lump in the throat." On the whole, however, I do not consider them the basic cause for this symptom. They are not a sign of the condition any more than is the symptom a disease entity. The sensation of the inability either to swallow or to bring up the lump is, to my mind, likened to the cramp one gets in a foot. I use this in explaining the sensation to my patients, most of whom come with the usual cancerphobia as a result of present day propaganda. In other words I would not consider it helpful to remove lymphoid material in these patients because it would fix the complaint in the direction of their phobic thinking.

During a recent visit to Hong Kong I was fortunate in seeing a number of cases of lateral pharyngitis as well as cancer of the nasopharynx. Dr. Douglas Laing, who gathered these cases together, said that both conditions were very common among the Chinese.

In his experience, lateral pharyngitis with inflamed ropey bands was secondary in most cases to a sinusitis, which is very prevalent in his part of the country. According to him, the chief complaints were intermittent clearing of the throat from the postnasal discharge and rawness or soreness of the throat. The sensation of a "lump" was not a predominant symptom.

If the patient still complained of a persistent soreness or discomfort in the pharynx after the sinusitis was controlled, Dr. Laing did not hesitate to remove the hypertrophied lateral bands surgically. In his hands this form of treatment has proved very satisfactory.

A number of local conditions have been described, which often produce abnormal sensations in the pharynx. Most of these lesions can be seen without difficulty by any competent laryngologist. Unfortunately, they can also be observed often by the patients themselves in a mirror, and, as a result, those with hypochondriacal natures frequently tend to worry.

Although some of the conditions which are often neglected have been mentioned, it is not intended to stress them to the exclusion of consideration of the upper entrance of the esophagus. This area automatically comes to mind when the term globus hystericus is mentioned.

When Sir Victor Negus [15] was questioned about his views on globus hystericus, he made the following remarks:

I have always associated the symptom of a lump in the throat either with lack of relaxation or excessive contraction of the cricopharyngeal sphincter, or with neoplasm in the pyriform fossa or post cricoid region. Occasionally I have noticed it in a case of enlarged and inflamed lingual tonsil, and once or twice with a mucous cyst in the vallecula; generally, however, I think the cause is near the mouth of the esophagus. I had not thought that lymphoid follicles on the posterior pharyngeal wall would cause it, as that usually gives rise to sensations in the pharynx, such as dryness or soreness. I have seen several cases of endothelial reticulosis, with large columns of lymphoid tissue on the posterior pharyngeal wall, but I do not think they cause globus hystericus. In most cases where the physiology of the cricopharyngeus is deranged I have found that dilatation of the sphincter with a hydrostatic bag will relieve the symptoms.

A recent article by Batson [16] is timely and gives a clear-cut description of this muscle:

The anatomy of the cricopharyngeus muscle may be summarized as follows. The fibers have their attachment to the cricoid cartilage laterally, at the junction of the annular portion with the "signet" portion of the cartilage. From these attachments

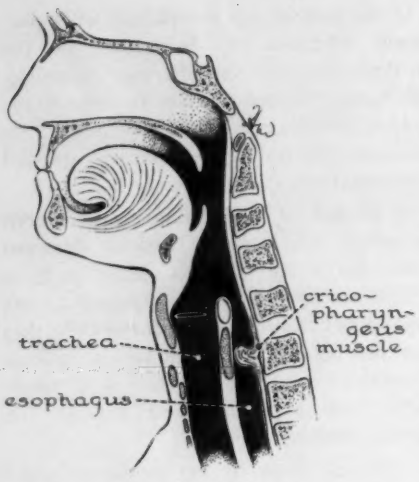

Figure 4

the fibers form a horizontal band-like sling which encloses the mucous membrane of the lower end of the pharynx. This is at the narrowing of the pharynx into the oesophagus. This band of fibers is 10 to 12 mm. high. Recall that the annular or horizontal fibers of the cricopharyngeus muscle have a very short total length as they course from the cartilage back to the cartilage around the entrance to the oesophagus. We have therefore an anatomic-physiologic mechanism which permits of a tremendous narrowing of the lumen by the short horizontal fibers which can contract to one-half of their length. This sphincter effect is increased since the anterior wall, the cartilage, is immobile.

Clinically Jackson [17] has stressed the importance of this sphincter muscle and calls it the cricopharyngeal pinchcock.

The cricopharyngeal pinchcock, as already mentioned, is produced by the tonic contraction of a specialized band of the orbicular fibers of the lowermost portion of the inferior pharyngeal constrictor muscle, called the cricopharyngeus muscle. As we have shown, it is this muscle and not the cricoid cartilage alone that causes the difficulty in the insertion of an oesophagoscope.

This constriction merits the name "cricopharyngeal pinchcock" because of the pinching shut of the oesophageal tube by an external muscle, whose action is similar to that of the pinchcock that pinches shut the rubber tube on the buret. Closure is tonic and constant; opening is co-ordinate and momentary, for deglutition and emesis. Even at complete relaxation a degree of structural narrowing exists. Saliva in small amounts goes through the closed pinchcock but any excess results in subconscious deglutition. The cricopharyngeal muscle relaxes at the same moment the inferior constrictor contracts, in normal swallowing.

In other words, the cricopharyngeus muscle is always in a state of tonic contracture even in the normal quiescent stage.

Negus has demonstrated that the purpose of this contraction is to prevent air from being inspired into the oesophagus, where it would serve no useful purpose and would only interfere with respiration by decreasing the effective tidal volume. Relaxation of the cricopharyngeus muscle occurs during deglutition, when the bolus of food stimulates the sensory fibers of the vagus as the bolus passes the pharyngeal constrictors. As the cricopharyngeus muscle relaxes, the larynx is closed and respiration temporarily ceases. During this relaxation phase the bolus of food passes from the cricopharyngeus area into the oesophagus, whereupon the cricopharyngeus muscle again constricts before respiration recommences.[18,19]

It is well to remember that the muscle is in a transitional location and has characteristics belonging in part to the constrictor of the pharynx and in part to the circular muscle of the esophagus. It has been established, as one might expect from the description, that the principal nerve supply to the cricopharyngeus muscle arises from the glossopharyngeal nerve and the inferior laryngeal nerve, a branch of the vagus. The tonicity of the muscle apparently varies from person to person.

These few anatomical facts are sufficient to show how proper muscular coordination is necessary in the act of swallowing. In normal well-adjusted persons deglutition is carried out harmoniously, without any discomfort whatsoever. However, in cases of nervous instability, when either real or imaginary tensions exist, many persons experience spasms or indefinite aches, and one of the commonest is the sensation of globus hystericus.

An attempt has been made in this article to list some of the common conditions which give rise to the sensation of a lump in the throat.

In addition, a few other lesions have also been mentioned, which sometimes cause this symptom but are often neglected by laryngologists.

A recent article by Mohun[20] describes some of the less common conditions.

In a comprehensive survey this year, Henry[21] reported his findings in 175 patients, over a five-year period, who complained of this symptom.

On questioning a patient, it is important to distinguish between difficulty in swallowing and the feeling of a lump. Difficult or painful swallowing is very suggestive of an organic lesion, while a "lump" is sometimes organic in origin but at other times functional. A complete history is, of course, important, as it often gives more information than the nose and throat examination. A thorough investigation of the nasopharynx, pharynx, and hypopharynx is necessary to rule out a local lesion as the cause of disturbed deglutition. In the absence of any positive findings to account for the disturbance in swallowing, the examination should be carried further, and this, of course, includes the esophagus and adjacent structures. In this way we are less likely to assume that the patient is suffering from some form of neurosis.

It is only by using all the means at our disposal that it is possible to arrive at a proper diagnosis and to institute the necessary form of treatment.

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The Effect of Cigarette Smoke on Ciliary Activity in the Upper Respiratory Tract

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The association between carcinoma of the bronchial tree and cigarette smoking has been increasingly discussed in recent years, and a voluminous literature has appeared on the subject. Concerning the immediate action of the smoke on the respiratory pathways, on the other hand, particularly in regard to mucus flow and ciliary beating, the literature is comparatively scanty. Proetz [1,2] demonstrated on animals that smoking produces a deposit of tar on the mucosa. In a series of three papers, Hilding [3-5] discussed the same question, but from more general as well as from specialized aspects. After studying the smoking habits of human subjects, he pointed out that these vary so widely that the number of cigarettes smoked is not a reliable measure of the smoke intake. The size of the smoke intake also differs very considerably according to whether the cigarette smoke is inhaled or only taken into the mouth.

Hilding further showed that in the space of a few seconds cigarette smoke could be so completely absorbed from the upper two-thirds of a cow's lung that scarcely any visible smoke remained. To study the effect of smoke on mucus flow he used tracheas removed from calves and cows. When cigarette smoke was passed through these tracheas mucus streaming ceased in 6 of 10 experiments. Observations of the actual ciliary beating were not possible in these experiments. To permit such observations, Hilding placed scrapings of epithelium from ciliated membrane in Ringer's solution and studied them in hanging drops under the microscope. Ciliary action was seen to cease when the epithelium was exposed to cigarette smoke. Hilding's experiments thus yielded extremely interesting results.

The effect of cigarette smoke on ciliary activity would seem to be the most pertinent aspect of the question under discussion, since slowing or cessation of ciliary beating implies direct consequences for mucus transportation. Observations of the mucus flow alone, however, scarcely permit binding conclusions in regard to ciliary activity. The mucus flow is also greatly influenced by changes in the composition and amount of the secretion. Such alterations may well result from cigarette smoking, as the ingredients of the smoke may conceivably either stimulate or depress the activity of the mucosal glands. By means of an apparatus which I have devised,[6] the frequency of ciliary beating can be calculated in living animals. With this apparatus I have studied the action of cigarette smoke on the cilia of the respiratory tract. The method was as follows.

Method

Ten live rats, ten extirpated tracheas from rabbits, and seven specimens of human mucosa, namely, four from maxillary or ethmoid sinus and three from bronchial tissue, were the materials in the experiments. The rats and the specimens were studied in a moist chamber with constant atmospheric temperature. For the experiments with the rats the chamber temperature was 34 to 35 C, and the rectal temperature of the rats averaged 36.8 C (98.2 F). For the extirpated specimens the mean temperature in the chamber and therefore also of the tissues was 36.6 C.

Smoke was introduced into the chamber in the following way. An assistant smoked a cigarette, collected the smoke in the mouth, and then slowly exhaled it through a short plastic tube over the

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TABLE 1.—*Effect of Cigarette Smoke on Rate of Ciliary Beat in Trachea of Living Rats*

Rat No.	Rate of Ciliary Beat (Beats/Min.) Initial Reading	After 5-Min. Exposure to Expired Air	After 1 Cigarette
1	1,034	1,063	747
2	1,108	1,121	Ceased
3	1,091	1,181	926
4	1,001	999	Ceased
5	1,470	1,258	Ceased
6	969	930	Ceased
7	1,329	1,109	Ceased
8	931	1,073	Ceased
9	1,122	1,040	832
10	935	890	Ceased
Mean	1,099	1,067	

extirpated specimens or the incised, exposed tracheas of the living rats, following their long axis. With a film camera the ciliary beating was registered immediately before the experiments began and also after a cigarette had been smoked for five minutes.*

To investigate if the *method* by which the smoke was blown over the cilia affected their activity, exhaled air without smoke was blown over tracheas at the same speed and for the same time (five minutes) as the smoke-containing air. The frequency of ciliary beat was then registered.

I have said that ciliary beating was registered by the method which I described in earlier publications. An improvement, however, was that the speed of the camera was increased from 128 to 220 exposures per second so as to permit more accurate counting of the films. The variations between individual rats as regards rate of ciliary beat and the error of method were studied by in vivo experiments on 10 rats under standard conditions.[6] The error of method was thereby found to be 35 beats per minute, and the interanimal variation was 162 beats per minute. The latter figure is not statistically different from the 176 beats per minute previously found.[6] The error of method, on the other hand, was significantly less ($P<0.001$ [xxx]) than the former value of 136 beats per minute. This appreciable reduction of the error of method (the counting error) was due to the increased camera speed. In consequence, it became possible to study the influence on ciliary activity of smaller effects than were earlier accessible to observation.

* Cinematographic registration was also made 20 minutes after the smoking ceased. The object was to see if effect would persist for some time after the exposure; in many cases the cilia did not seem to recover. It must be stressed, however, that effective removal of the air in the moist chamber was not technically feasible, one reason being the necessity of keeping the temperature constant. It is thus possible that the concentration of smoke remaining in the chamber was sufficient to maintain the original effect. These findings after 20 minutes cannot therefore be directly applied to normal circumstances.

TABLE 2.—*Effect of Cigarette Smoke on Rate of Ciliary Beat in Extirpated Rabbit Tracheas*

Specimen No.	Rate of Ciliary Beat (Beats/Min.) Initial Reading	After 5-Min. Exposure to Expired Air	After 1 Cigarette
1	1,156	966	Ceased
2	1,347	1,000	1,121
3	901	909	940
4	1,021	999	Ceased
5	847	840	Ceased
Mean	1,054	943	
6	997	No test	Ceased
7	1,206	No test	Ceased
8	1,164	No test	Ceased
9	805	No test	837
10	967	No test	1,173

Results

The results of the experiments with cigarette smoke are presented in Tables 1, 2, and 3. As regards Table 1 (observations on rats), no statistically significant difference was found between the mean ciliary beat rate at the start of the observations (1,099 beats per minute) and that after exposure to a flow of expired air for five minutes (1,067 beats per minute). In 7 of the 10 rats, ciliary movement ceased after exposure for five minutes to cigarette smoke.

The findings with extirpated rabbit tracheas are shown in Table 2. Measurements after exposure to a flow of air were made

TABLE 3.—*Effect of Cigarette Smoke on Rate of Ciliary Beat in Extirpated Human Mucosa*

Specimen	Beats/Min. Initial Reading	After 1 Cigarette	Chamber Temperature, C
Maxillary sinus	818	Ceased	36.9
	740	Ceased	37.6
	676	Ceased	36.5
Ethmoid sinus	995	Ceased	36.9
Bronchus	969	Ceased	37.3
	823	Ceased	37.0
	854	686	37.2

on the first five of the specimens. The difference between the preceding mean rate, 1,054 beats per minute, and the subsequent mean, 943 beats per minute, was not statistically significant. It nevertheless implied some tendency to deceleration, probably because the specimens were examined in vitro. Neither in the present series nor in the previous studies was ciliary movement in live rats affected by a flow of air. Extirpated tissues, on the other hand, were found in earlier experiments to compare unfavorably with in vivo mucosa as regards stability of both mucous streaming and ciliary activity.

Of the 10 tracheas exposed to cigarette smoke, 6 reacted with complete cessation of ciliary beating. In the other four specimens no certain effect was observed.

The observations on the seven tissue specimens from human subjects are reviewed in Table 3. In six cases the ciliary movement ceased after exposure to tobacco smoke. In the other specimen (from a bronchus), the cilia continued beating but the rate fell from 854 to 686 beats per minute, i. e., by 168 beats.

Comment

The immediate effect of cigarette smoke on the ciliated mucosa of the respiratory pathways in these experiments was that in most cases the ciliary activity ceased. Mucosa from different experimental animals and from human subjects displayed similar reactions. The cause must be regarded as action on the ciliary mechanism by ingredients of the smoke which were dissolved in the mucus layer. As the concentration of smoke could not be graduated with the method used, it cannot be excluded that lower smoke concentrations would have produced a milder effect. In smokers who inhale, however, one may assume that the concentration of smoke in the trachea is not appreciably lower than that in my experiments. Cessation of ciliary activity entails stagnation of mucus secretion. How far down in the respiratory pathways such a reaction extends cannot be judged at present.

From the foregoing it appears that cigarette smoke contains substances which, after dissolution in the mucus layer, influence ciliary movement so that, under the conditions of the experiments, it ceases after, at any rate, five minutes of exposure to the smoke. The similarity of reaction in different animals and the fact that the concentrations of smoke presumably did not exceed those reached in human subjects who inhale cigarette smoke imply that ordinary smoking habits may result in the described effect on ciliary movement at least in the upper part of the respiratory tract. Whether or not this circumstance has any relevance to the concomitant increase of cigarette smoking and bronchogenic cancer was not within the scope of the investigations herein described. However, I have planned experiments with single and with repeated exposure to cigarette smoke in which the ciliary movement in the trachea can be continuously observed with a special apparatus while the animal itself "smokes" a cigarette. The actual smoking will thereby be more physiologic, as the natural absorption mechanism of the respiratory pathways will come into play.

Summary

The frequency of ciliary beating was registered in the trachea of live rats and in extirpated rabbit tracheas and specimens of human sinus and bronchial mucosa. After exposure to cigarette smoke for five minutes ciliary movement ceased in 18 of the 25 experiments.

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Effect of Conventional Fenestration on Ménière's Disease

Consideration of Some Other Operative Methods

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The literature on Ménière's disease has swelled to the extent that not even the most remarkable papers can be reported in a short article. A very complete list is found in Altmann's exhaustive review (1955), which deals with the relevant problems and considers them from every conceivable angle. Therefore, only a few reports that have since been published will be mentioned here.

At present the general conception of the pathology of the disease is in agreement with the opinion of Hallpike and his collaborators, who contend that increased pressure within the membranous labyrinth is the characteristic feature. However, the etiology and the pathogenesis are still obscure. From some experiments and observations a conclusion has been drawn according to which a spasm of the arterioles in the internal ear would cause edema. This must also influence the endolymphatic and perhaps also the perilymphatic secretion, producing a disturbance in this function. A simultaneous disturbance of the resorption of these fluids must then exist. Our knowledge of the action of the corresponding nerves is still deficient. There is no fixed conception of the changes in the organ of Corti and the sensulae in the vestibular organ. Since all the problems mentioned and many other relevant questions are still unsolved, it seems desirable to report observations made in different materials. They may, even if not decisively, contribute to a solution of some of these problems.

Since an increased pressure prevails in the labyrinth of a Ménière patient, it is natural that decompressing operative procedures have been suggested and undertaken, especially in cases that have not responded to conservative medical treatment. As early as 1951, I reported favorable results in patients treated with a simple fenestration of the kind used in otosclerosis. As the series has now increased and we have been able to investigate the patients more closely, publication of our observations seems indicated not so much for the purpose of advocating this operation, though it has been beneficial in numerous cases, but to add to our knowledge of the behavior of the disease.

It has been claimed (Altmann, Lindsay, Harrison) that a fenestration of the external canal in Ménière's disease gives unsatisfactory results. Our material proves that in the great majority of cases there has been a considerable change in the condition of the patients. I therefore feel that our material is worth scrutinizing. Originally this procedure was proposed by me with the object of preserving the function of the cochlea. However it must be admitted that the operation did not meet our expectations in this respect. There must exist a special factor in the internal ear of a Ménière patient which acts unfavorably on the hearing sensulae. This has also been observed by others, but authors seem to differ in their interpretation of the nature of the phenomenon. The Portmann operation which has the same decompressing aim, although applied directly to the endolymph, also has a similar effect upon the hearing. These observations will be discussed later.

A possible role of the action of sympathetic or parasympathetic nerves upon the

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secretion of labyrinthine fluids has been much discussed. According to some observations the exclusion of the sympathetic trunk should lower the excitability of the labyrinth. This has induced therapeutic measures attacking the sympathetic, such as blocking and extirpation of the stellate ganglion and extirpation of the upper thoracic ganglions. Passe has performed numerous extirpations of the second and third thoracic ganglia and has claimed good results on Ménière's disease. Dix and Harrison also report cases in which this operation was done, although they are not so enthusiastic as Passe. Our series of Ménière patients operated on include five treated by this operation; they will also be reported below.

Vertiginous Attacks

By far the largest group in the present material consists of those patients on whom a simple fenestration was performed. This group will be dealt with first. The most important aim of therapy in Ménière's disease is to relieve the patient of the incapacitating and dreaded attacks of vertigo. The following table illustrates the results in this respect. It contains observations on 56 cases, during a follow-up time varying from three and one-half months to more than nine years. It is interesting to note that about 79% of the patients operated on were freed of typical attacks of vertigo. Thus, a procedure which hardly touches the membranous labyrinth had an effect on its function.

TABLE 1.—*Effect of Fenestration on Attacks of Vertigo*

Follow-Up Time, Yr.	Patients, No.	No Attacks	Attacks, No.
3½-10 mo.	5	3	2
1-1 11/12	8	5	3
2-2 10/12	2	2	—
3-3 11/12	6	4	2
4-4 8/12	6	6	—
5-5 6/12	6	5	1
6-6 8/12	2	2	—
7-7 11/12	10	9	1
8-8 11/12	9	6	3
9-9 9/12	2	2	—
Total	56	44 (78.57%)	12 (21.43%)

The attacks, with rotation of surrounding objects, subsided, and most of the patients were able to resume their ordinary work. Eight of them stated that sometimes but very rarely they experience slight giddiness, lasting in a few cases as long as 10-15 minutes. There were two patients who felt giddy for a short time when overstrained. One woman had a similar sensation a couple of times at the beginning of a pregnancy. One patient could not lie on the side of the ear operated on but had no sensation of giddiness in other postures. A man, over 50 years of age, complained of a continuous feeling of unsteadiness. He had been suffering from high blood pressure for several years. As he is now free from his vertiginous attacks, which preoperatively were frequent, it seems justified to count him among the cured.

From Table 1 it appears that 12 patients continued to suffer from attacks of vertigo after the operation. The intensity and the frequency of the attacks vary considerably. In many of these cases the condition had clearly changed after the operation. As the attacks have shown the typical illusional turning of objects, these patients were included in the group with persisting attacks, although some of them really belong to the group who benefited from the operation. It therefore seems advisable to describe some of these cases briefly.

Report of Cases

The first group, much improved or almost cured, includes three patients.

CASE 1.—A woman was operated on by fenestration at the age of 54 years. About one-half year after the operation she had had several attacks of vertigo, considerably milder than preoperatively, for three months. Then followed an attack-free period of eight years. She then occasionally had a brief sensation of giddiness without illusional turning of objects. When examined later she was again free of any sensation of giddiness.

CASE 2.—A woman, who was 47 years of age at the time of fenestration, when traveling in a bus after the operation had an attack of vertigo which lasted one hour. Eight months postoperatively she had an attack lasting two minutes, during

which objects seemed to turn. When examined seven and one-half years after the operation she had been totally free from attacks. The patient suffers from high blood pressure.

CASE 3.—A woman whose left ear had been fenestrated when she was 33 years old, had two attacks of vertigo two months after the operation and three attacks one year postoperatively, at the beginning of a pregnancy. Since then, for two and one-half years, she has been completely free from attacks.

The second group also consists of three patients.

Two were women who had had fenestrations done, one 10 months previously and the other 22 months previously. Both were reoperated on, with destruction of the labyrinth because attacks had recurred. The third was a man, 24 years of age, who after fenestration experienced three faint attacks of vertigo but urged reoperation because of a constant feeling of unsteadiness. The labyrinth was destroyed.

The third group consists of six patients with typical attacks.

Only one has severe and frequent attacks. Two of the patients have been free from attacks for the last two postoperative years. Three patients report that their attacks are obviously weaker than before fenestration.

Of the 12 patients, the condition of the first 3 has improved so much that they actually are much nearer the group of cured than the group of those not cured. If they are added to the 44 attack-free patients, the number of those benefited is 47 of the total 56, or 83.92%

From the results it may be concluded that the operation obviously affected the disease and most of the patients benefited.

If the five cases (Table 1) observed for only 10 months are omitted, the corresponding figures are 51 fenestrations; in 41 of these cases freedom from attacks resulted, but in 10 attacks persisted. Thus 80.3% were relieved of vertigo. If the three patients who were actually healed, although they experienced a few occasional attacks, are added to the cured, the number of cured thus rises to 44, the per cent of positive results being 86.74%.

Condition of Fenestra and Attacks of Vertigo

It is of interest to know whether patency of the fenestra is a prerequisite for freedom from attacks of vertigo. A positive fistula test confirms the patency while a negative one does not always assure a closing. This is illustrated in Table 2.

TABLE 2.—*Patency of Fenestra and Attacks of Vertigo*

Fistula Test	Cases No.	No Attacks of Vertigo, No.	Attacks of Vertigo	No Notice
Positive, regular	16	12	1	3
Positive, irregular	6	5	1 (a few, slight)	
Positive, weak	10	8	2 (1 slight)	
Negative	13	10	3 (2 slight)	
Total	45	35	7 (4 slight)	3

Thus, only one of those who had a regular fistula test suffered from attacks of vertigo similar to those before the operation. The six patients with a positive irregular fistula test were almost all free from attacks. The only one who reported attacks was a woman who had had a few attacks shortly after the fenestration but none for the past seven years. The attacks had been very frequent before operation. The irregular response to the fistula test in these cases was mostly a nystagmus directed to the same side by both pressure and aspiration. Of the 10 patients showing a positive but weak fistula symptom, 2 reported attacks. One of them had suffered from typical episodes for four years postoperatively but had now been free from them for more than three years. The other, a woman of 37 years, had had a couple of slight attacks a short time after the fenestration but none for the past three years.

In 13 cases the fistula symptom could not be elicited. Ten patients had been quite free from attacks of vertigo, the follow-up time varying from 9 years 3 months to 3 years. One patient, 43 years of age, had experienced a few attacks in strenuous work, and another one, of the same age, had suffered from attacks, particularly in the mornings, but they had been much weaker than before the fenestration. One woman, 46 years of age, had suffered from frequent

TABLE 3.—*Positive Postoperative Caloric Reaction in Thirty-Three Patients* *

Caloric Reaction				Rot. Test, Duration		First Test	
Equally Strong on Both Sides	Weaker on Fenestrated Side	Nystagmus Reversible When Head Bent Down	Nystagmus Not Reversible	Equal	Shorter	Positive	Negative
13	19	6	18	16	17	21	5

* No attacks, 26; mild attacks, 4; severe attacks, 3.

slight episodes of vertigo until the labyrinth was destroyed by coagulation.

Table 2 shows that most of the patients with a negative postoperative fistula test were also relieved of vertigo. Why the fenestration was successful although the fenestra closed is not easy to say. It is improbable that the wall of the membranous canal would have been damaged in the majority of these cases.

The patients were also asked about dizziness on rapid movements of the head. Twenty replied affirmatively, while eight had not experienced any dizziness. It is surprising that these latter included only one with a negative fistula test.

Caloric and Rotation Tests

Y. Meurman and Pursiainen earlier examined fenestrated otosclerotic patients by caloric and rotation tests. In many respects the results agreed with those in the present material.

Method

Forty-seven patients were available for these examinations. The caloric test was made with air at 20-22 C, blown through an ear speculum with a thin metal tube against the drum for one minute, after which the duration was noted. In the rotation test, the subject was turned 20 times in 20 seconds, and the duration of postrotatory nystagmus was timed with a stop watch. The subjects were divided into two groups: those with a positive caloric reaction and those with a negative one on testing. Of these patients, five were operated on behind the ear and fenestra was made in the middle of the canal wall, without removal of the incus. Four patients had suffered from slight postoperative attacks, and three had had severe attacks. All of the other patients were free from vertigo.

Results

No correlation was found between the appearance of the postoperative attacks of vertigo and the weaker caloric reaction on the side operated on. Nor was there any correlation between the shorter postrotatory nystagmus and the attacks of vertigo. Table 4 also includes three patients operated on behind the ear who consequently could not be examined by the fistula test. Only two patients had had severe postoperative attacks of vertigo.

In the group with a positive caloric reaction, about one-fourth of the patients had had postoperative attacks of vertigo, while, in the group with a negative reaction, more than one-third (5 of 14) had had such attacks. As the latter group is much smaller, I am not sure that the result is significant. Of the five patients with attacks of vertigo, three had a negative and two had a positive fistula symptom.

Tinnitus

To an inquiry about tinnitus I received replies from 30 of the fenestrated patients. Of these, 29 suffered from murmur or buzzing of varying intensity. In many cases there was also a superimposed or alternating

TABLE 4.—*Negative Postoperative Caloric Reaction in Fourteen Patients*

Rotation Test, Duration					
Equal on Both Sides	Shorter on Side Operated On	Fistula Test		No Attacks of Vertigo	Attacks of Vertigo
		Positive	Negative		
7	7	7	5	9	5

higher sound, a kind of ringing or chirping. Only one reported continuous ringing. The murmur was markedly weak in nine cases and strong or distressing in four cases. The others apparently tolerated the tinnitus without noticeable distress. Obviously a simple fenestration does not in general relieve the patient of tinnitus. Comparison of these patients with those whose membranous labyrinth was destroyed shows that even of these none was entirely relieved of tinnitus. In only one case was the murmur in the ear operated on very weak.

Hearing

It is a striking and very surprising observation that in many cases hearing disappears or is considerably impaired in the affected ear of a Ménière patient within a relatively short time after an ordinary fenestration. However, there are exceptions, in which hearing has remained on approximately the same level for a long time postoperatively. Our material includes a large number of patients who were already preoperatively deaf in the affected ear. In these cases, naturally, no conclusions regarding the acoustic function can be made. Those with considerably deteriorated hearing before fenestration—perhaps the majority of these patients—are not likely to give a clear picture of a possible change. Nevertheless, many of these illustrate to some extent postoperative changes in the acoustical function. The following list gives an account of changes in hearing after the fenestration:

Change in Hearing	Cases, No.
Hearing lost	13
Much impaired	9
Distinctly impaired	2
Slightly impaired	11
Scarely impaired	1
Unchanged	3
Total	41

This list shows that in about one-third of the cases the hearing disappeared and that further impairment took place in four-fifths of the rest. In only about 10% hearing remains on the preoperative level. This record

TABLE 5.—*Change of Hearing Within One Month Postoperatively*

Groups	Cases, No.
Hearing entirely lost	10
Minimal residual hearing	5
Conversation speech impaired to mostly a. c., 0.2 m.	7
Hearing slightly impaired	1
Hearing as before fenestration	2
Slightly improved	1
Total	26

does not, of course, clearly illustrate the postoperative changes. To the character of the disease itself belongs a continuous diminution of hearing. Regular examination of hearing at fixed intervals postoperatively would elucidate the problem much better, but it was not possible to carry out such an examination. However, 26 patients were examined within one month after the fenestration. The influence of the operation is illustrated in Table 5. In more than half of the cases hearing was either entirely lost or severely impaired. In the group with slight impairment the preoperative level had already been low and the change was thus small. About 10% of these patients (three) did not lose any of their hearing capacity. Thus it is not to be denied that this procedure, although outside the membranous labyrinth, not to speak of the cochlea, undoubtedly seems to have a damaging effect on cochlear function in many cases.

In trying to find an explanation we may ask whether the site of the new fenestra may have some influence in this respect. In this material there are eight cases in which the fenestra was made farther back in the canal wall. In every case the fenestra was covered with amnion membrane. As the incus was not removed in these cases, one might expect that hearing would not be influenced noticeably. In three patients the affected ear was totally deaf before the operation. There remained five patients with some hearing. Two of these had the best hearing: conversational speech at a distance of 0.2-0.7 meter. All five became totally deaf in

the ear operated on, two of them within four months. From these cases we can conclude that the site of the new fenestra, farther from the ampulla, does not reduce the tendency to postoperative deterioration of hearing.

As already pointed out, a fenestration was generally performed only on ears with relatively severely impaired hearing. However, those operated on included three with less hearing loss: one heard conversational speech at 1-2 meters and one at 2-4 meters. The first mentioned was evamined 11 months after the fenestration and then heard only part of the words *ad concham*. The second patient hardly heard with the fenestrated ear any conversation speech *ad concham* 46 days postoperatively. At the follow-up examination the first had been free from attacks of vertigo for more than nine years, the second for three years seven months. The third patient heard conversation at 2-5 meters with the affected ear. One year five months after operation, hearing in this ear was 3-6 meters. We can say, at any rate, that it had not been impaired; rather it had improved, as shown also by the audiogram. However, nine years two months after the fenestration the hearing distance for conversation had diminished to 0.2-2 meters. This also corresponded with the audiogram. The patient had for the whole postoperative period been free from attacks. Although this is an exception, it shows that there may be cases in which a fenestration does not damage the hearing capacity. The hearing loss after nine years may be due to the disease itself and independent of the fenestration. We have here marked the hearing for conversational speech with two figures. The lower figure expresses the hearing for words with low frequencies, the higher figure for high-frequency words.

We may ask what effect the removal of the incus has on hearing in these cases. We generally accept as a fact that it lowers the AC graph 20-30 db. In cases with conversational hearing at 2-4 meters, this would probably be expected also in a Ménière ear. But, if hearing is already considerably depressed, let us assume, for instance, to 0.2-0.5 meters, removal of the incus probably has much less effect. One ear with the preoperative hearing at 2-4 meters lost it entirely in 20 days. Here removal of the incus could not have been the cause. In this case, the audiogram taken 12 days postoperatively showed a slight lowering of the BC graph and a considerable lowering of the AC graph (500 cps, 50 db.; 1,000 cps, 40 db.; 2,000 cps, 15 db.). Six days later the BC graph was lowered about 10 db. and could not be traced higher than at 2,000 cps, but at the lowest point (1,000 cps) it was still on a 60 db. level. The initial hearing of another patient was 1-2 meters in the affected ear. At operation, after removal of the incus, hearing did improve slightly, up to 3 meters. However, 17 days after operation speech was heard only from *ad concham* up to 10 cm. The BC graph was lowered 15 db. only at the high frequencies (2,000-4,000 cps); the AC graph was lowered 10 db. at 250 cps., 20 db. at 500 cps, 60 db. at 1,000-2,000 cps, and 15 db. at 4,000 cps. This case proves (test at operation) that removal of the incus need not reduce the hearing capacity, at least in cases with moderate hearing impairment. Later the ear lost hearing for speech.

Like O.H. Meurman and Grahne, I have examined the AC audiograms in 52 Ménière cases. Among these were 12 cases with rising graphs, 31 cases with flat graphs, 6 with gradually falling graphs, one with a graph with a dip, and 2 with an abruptly falling graph. The postoperative course in the audiograms could be followed in 31 cases. The graph initially improved in three cases; it remained at the same level for a long time in two cases and for a shorter time in five cases. In 11 cases the audiograms showed a slow gradual impairment, and in 10 cases the impairment took place more rapidly. Thus, the course was in accord with the results obtained from examination of the hearing by conversation and whisper.

I tried to check the prognosis of hearing in the different audiogram groups. However, no definite regularity was found. The graph was best maintained in the group with gradually falling curves; two kept on the same level for a long time and one for a shorter time, while one was slowly impaired. The whole group consisted of six cases, of which only four could be followed up.

O. H. Meurman and Grahne have expressed the opinion that the postoperative hearing loss in fenestrated Ménière ears is due chiefly to an aseptic labyrinthitis. Lindsay has also mentioned the same explanation. It seems not probable that this is the main cause. Firstly, postoperative nystagmus in these cases did not last longer on an average than in cases of fenestrated otosclerosis. Secondly, in cases in which hearing was totally lost within a short time, the duration of postoperative nystagmus was not longer than in cases without total postoperative hearing loss or in cases in which hearing remained on the preoperative level (e. g., 481/54 for six days, 981/49 for four days, 329/49 and 1,223/54, 854/50 for no days). Thus, at least, an aseptic postoperative labyrinthitis cannot alone account for the hearing loss in these cases. We have checked all the fenestrated-ear cases suffering from otosclerosis since 1953 (44 patients). Although there were patients who were entirely free from postoperative nystagmus, there were some who showed it for as long as two to three weeks. On an average, it lasted five days. Even those with prolonged nystagmus did not lose their hearing improvement. Consequently, it would seem that there must be some other factor—a specific one causing the deterioration.

An example is the case of a middle-aged man who suffered from a very severe tinnitus in one ear. Various remedies were tried, without result. Finally, by way of an experiment, it was decided to perform a fenestration on that ear. The incus was left. There was no effect whatever on the tinnitus. The hearing, however, remained on the preoperative level.

TABLE 6.—*Hearing Totally Lost Postoperatively, Eighteen Observations*

Examination Time Postoperative	Cases, No.	Postoperative Nystagmus No.	Grade	Duration of Nystagmus No.	Days
6- 8 days	1	1	III		6
11-19 days	3	2	III		4
		1	II		1
20-28 days	4	4	II	1	1
				3	?
				1	2
30-37 days	3	3	II	1	4
				1	5
46 days	1	1	III		3
6 mo.	2	1	III		?
3 mo.		1	I		5
1 yr.	1 *	1	II		6
4 yr. 8 mo.	1 †	1	III		5
7 yr. 6 mo.	2	2	II	1 ‡	5
				1	9
Total	18	Range, I-III		Range, 1-9	

* When examined 1 mo. after operation, hearing was on preoperative level.

† When examined 7 days postoperatively, hearing showed very slight further loss.

‡ When examined 1 mo. postoperatively, hearing was very slightly deteriorated.

The hearing was checked postoperatively at intervals of varying length, and the degree of deterioration was noted. Altogether 44 such observations were made, not including patients who were totally deaf before operation. The cases were divided into four groups: I, hearing totally lost; II, hearing much deteriorated from preoperative level; III, hearing slightly deteriorated from preoperative level, and IV, hearing maintained on preoperative level. Tables 5 and 6 illustrate the relation of postoperative nystagmus and its duration to the degree of hearing impairment.

Of these patients, six had Grade III postoperative nystagmus; 11 had Grade II nystagmus, and 1 had Grade I nystagmus. It should be noted that one of these patients showed only Grade I nystagmus for five days and two Grade II nystagmus for one day.

In the group with severely deteriorated hearing, 10 patients were examined within one and one-half months after operation. Three of them initially had Grade III nystagmus; five had Grade II nystagmus, and one had Grade I nystagmus. In the

TABLE 7.—*Intensity and Average Duration of Postoperative Nystagmus* *

Group	Total Cases	Grades of Postoperative Nystagmus III	II	I	Mean Postoperative Duration, Days	Patients, No.
1	12	4	8	—	3.75	8
2	10	3	6	1	4.66	6
3	7	1	3	3	5	6
4	3	—	2	1	4.7	3
Total	32	8	19	5	4.53	23

* I indicates hearing totally lost; II, hearing much deteriorated; III, hearing slightly deteriorated; IV, hearing maintained on preoperative level.

group of seven patients with slight further loss of hearing, examined within the same postoperative period, only one had Grade III nystagmus, three had Grade II nystagmus, and three had Grade I nystagmus.

The group with maintained preoperative hearing after the fenestration consisted of three patients examined within the period mentioned. Two of these showed Grade II nystagmus, lasting five and six days, respectively, and one showed Grade I nystagmus for three postoperative days. This patient, examined 13 months postoperatively, had retained the preoperative hearing.

Table 7 shows the distribution of the various postoperative nystagmus grades among the groups and the average duration of postoperative nystagmus. On studying this Table one must admit that the number of patients presenting a Grade III postoperative nystagmus was greater in Groups 1 and 2 in whom the hearing was totally lost or much deteriorated postoperatively, than in Groups 3 and 4, who had only slightly deteriorated or unchanged hearing. This supports the opinion that irritation in the labyrinth or perhaps "aseptic labyrinthitis" may be a factor in the deterioration. On the other hand, as observed from Table 6, there are cases in which the irritation was sometimes very weak, with Grade II nystagmus for only one day (two cases), and yet the patients became deaf.

From the results of this detailed survey we may conclude that although a postoperative irritation, perhaps "aseptic labyrinthitis," seems to be one cause of postoperative hearing deterioration, there must be one or more additional factors.

Retroauricular Fenestration with Preservation of Incus

In nine cases the fenestra was opened on the vertex of the horizontal semicircular canal, with use of the same technique as in the usual site. It was then covered with fresh amnion membrane obtained from the Obstetrical Hospital. The incision behind the ear was sutured immediately.

Among these cases were three patients totally deaf before the fenestration. It may be noted here that these patients nevertheless suffered from severe attacks of vertigo preoperatively. Thus it is additional evidence that a total hearing loss in Ménière's disease does not ensure subsidence of attacks. Two of these patients got rid of attacks after the fenestration, but one did not, though the attacks became considerably weaker.

Of the six patients not deafened, five became free from attacks of vertigo. One suffered from attacks postoperatively, but they were much less frequent.

With use of the above technique the postoperative reaction of the labyrinth would have been expected to be extremely slight, a possibly deleterious influence on the hearing organ being avoided. Preservation of the incus ought to have contributed to the preservation of hearing too.

Immediate postoperative nystagmus appeared in seven patients. Two patients showed no nystagmus immediately after the operation, but later there was nystagmus, though slight, lasting 15 to 18 days postoperatively. These two patients were already deaf in the ear operated on before the fenestration. In the case of one patient data are lacking. Only one of these patients had a Grade III nystagmus immediately after the operation; three showed a Grade II nystagmus, and three, a Grade I nystagmus.

The duration of the nystagmus also did not generally differ from the one usual in otosclerosis patients after fenestration. It varied between seven and three days in all except the two patients already mentioned.

On reexamination of the six patients whose ears operated on had not lost their hearing capacity preoperatively, one showed total hearing loss at 15 days and three at four months; one patient discerned loud words *ad concham* at 2 months, but hearing was later totally lost. One ear was not reexamined until one year postoperatively and was then totally deaf.

This survey of the results of fenstrations performed on the vertex of the canal shows that the postoperative reaction of the labyrinth does not differ from that appearing in patients with fenestrations done in the usual manner, close to the ampulla. Nor does this technique in any way ensure a preservation of the preoperative hearing levels.

Working Capacity

Not only freedom from attacks of vertigo but also restoration of working capacity is of vital importance for the patients. This problem, however, offers many difficulties. During the period of attacks the patients generally were more or less incapable of doing their ordinary work—and some of them not even during the intervals between attacks. The length of the disease varied greatly and so the working capacities of different patients before the onset of the disease can hardly be comparable. In addition many of the sufferers were of advanced age, a fact that influenced their fitness for work. In our questionnaires we asked only about their postoperative working capacity, and it is scarcely probable, for the reasons stated above, that the replies even to a more precise question would have been more informative than those obtained.

We received valuable answers from 32 patients on whom a simple fenestration had been performed. Fourteen of those operated on were fully fit for work. Working capacity was slightly diminished in the case of six patients. Nine patients who had fenestrations complained of distinctly reduced working capacity, and one said that it was much impaired. Two were incapable of work: One suffered from cancer of the lung and the other from high blood pressure.

How much the age and the condition of the nervous system influences this burden is difficult to know. Other figures give no reliable answers. The age in the groups with some complaints varied between 49 and 53 years, whereas those with a good working capacity averaged 50.8 years.

In conclusion it may be said that about two-thirds were restored to fairly good working ability. Comparison with other materials is hardly feasible. In the present work the average age of these patients was so high that the working capacity may have been influenced by many other factors.

Destruction of the Membranous Labyrinth

In six cases the membranous labyrinth of the diseased ear was destroyed; in three cases this was performed primarily. The indications in this series were not consistent, and these operations were not done by me. In the three remaining cases the destruction was made secondarily. All these cases will be briefly described for comparison. The primarily destroyed labyrinths are presented first.

Case 1.—A woman, 50 years of age, had suffered from attacks of vertigo for four years, and during the last four weeks they had returned daily. The affected ear was completely deaf. The horizontal membranous semicircular canal and a part of the utricle were removed. During follow-up, two years and five months later, the patient felt completely well; neither attacks nor giddiness occurred. No sensation of dizziness on head movements was felt. Slight uncertainty of gait was experienced when she walked in the dark. There was a weak murmur in the ear operated on. Working capacity was good.

Case 2.—A woman, aged 51 years, heard with the diseased ear conversation at a distance up to 0.4 meters. She was first given a few injections of procaine into the stellate ganglion and was then soon operated on. The membranous horizontal semicircular canal, together with the ampulla, was removed by suction. The attacks of vertigo from

which she had suffered for 14 years immediately ceased. No giddiness was felt on head movements; the equilibrium was faultless. The patient was free from attacks and giddiness for the whole follow-up period of three years six months. Her only complaint was faint ringing in the ear when fatigued. Working capacity was good. The fistula test was negative.

CASE 3.—A woman, aged 45 years, had suffered for eight years from murmur and occasional ringing in the right ear. Attacks of vertigo lasting two to three hours had occurred about once a month during the last two years. During the last three months they had become more frequent, generally once a week. Conversational speech was heard partially at to 20 cm. A part of the membranous horizontal semicircular canal was removed at operation. The patient was followed for five years and ten months. She had no attacks of vertigo until four and one-half years after the operation. However, the operation cavity was then filled with epidermal scales. When these were removed, the attacks did not recur. Working capacity was slightly impaired.

Comment.—In three cases the membranous labyrinth was destroyed some time after simple fenestration, which evidently had not been fully successful:

CASE 4.—A woman, aged 48 years, had suffered from attacks for 10 months, lately once a month. The diseased ear heard the conversational voice at a distance of 0.3-1.3 meters. A simple fenestration was performed. The attacks of vertigo recurred after four months and were very frequent, often daily. As the condition did not improve, the labyrinth was reopened 10 months later, the membranous semicircular canal extirpated, and the utricle coagulated. After this the attacks were absent for a couple of years, but they recurred, though weaker and at longer intervals. A murmur was heard in both ears. The possibility of the attacks having their origin in the ear not operated on could not be entirely excluded. The total observation time exceeded nine years.

Case 5.—A woman, aged 39 years, had suffered from attacks for two years. A simple fenestration was performed in May, 1950. In October, the attacks of vertigo recurred; one of them lasted as long as two hours. Attacks then began to occur daily, lasting 10 minutes. Refenestration was made in the middle of December, 1951. At the site of the previous fenestra two tiny openings in the bone were found. The membranous canal behind the ampulla was totally obliterated. The utricle was coagulated. Attacks have not recurred, but the patient suffers from dizziness. There is a loud murmur in the ear. She has been followed six and one-half years after the second operation.

CASE 6.—A man, 24 years of age, had suffered from attacks of vertigo for one and one-half years. A simple fenestration was performed in September, 1954. After that he had only three slight attacks of vertigo, but there was constant equilibrial uncertainty. Murmur in the ear had also increased. He was anxious to have a reoperation. This was performed on Feb. 2, 1955. The fenestra was reopened and widened in the anterior direction. The horizontal and a part of the vertical membranous semicircular canal and the utricle were extirpated with forceps. The postoperative observation time is three years. The patient sometimes feels giddy for 15 minutes, but he has had no attacks of vertigo. He is fully capable of working. Equilibrium is, on the whole, good. He suffers to some extent from murmur in the ear, which has increased after the operation.

Comment.—After the destruction of the membranous horizontal semicircular canal and a part of the utricle all these patients were relieved of attacks of vertigo. Movements of the head did not cause giddiness. However, some patients occasionally felt dizzy, but this generally did not affect their working capacity. No one was relieved of tinnitus, which had the character of murmur in all cases except one, who had faint ringing in the ear.

Sympathectomy

As operations on the labyrinth, even a simple fenestration, seemed to have a deteriorating effect on the hearing in Ménière cases, it was tempting to try an operative method outside the internal ear. The thoracic sympathectomy of Passe, which according to its inventor gave very good results, seemed suitable. Five patients were submitted to this operation. We extirpated the second and third thoracic ganglion, together with the sympathetic trunk between them, and a portion of the joining intercostal nerves. The cases will be briefly presented. Cases with relatively good hearing in the diseased ear were particularly suited for this procedure.

CASE 1.—A woman, 28 years of age, had suffered from murmur in the ear and slight attacks of vertigo for more than two years. The interval varied from one day to some months. Occasionally there were several attacks on the same day. Hearing for conversation ranged from 0.8 to

5 meters, for whisper from 0.2 to 1 meter. A right thoracic sympathectomy was made in February, 1952. After the operation, the right upper extremity was warmer than the left. The patient was examined in October, 1956 (four years and seven months postoperatively) because she was still suffering from occasional attacks of vertigo. Hearing had become distinctly impaired, being 0.3-0.4 meters for conversation.

The patient reappeared for examination in September, 1958. From November, 1957, to Sept. 1, 1958, there were neither attacks nor giddiness, but on the day she was examined she had slight vertigo for a few minutes. Buzzing in the ear continued. The right hand was drier than the left and occasionally colder. Reading was sometimes difficult. The caloric reaction was of equal strength on both sides. Conversational speech was heard at 0.3-0.8 meters and whisper at 0-*ad consham*. The audiogram was similar to that taken in 1956.

Case 2.—A man, aged 37, had hit his head in an accident, in December, 1949. His left temporal bone was fractured, and he lost consciousness for 15 hours. In November, 1950, his right ear was blocked, and he began to suffer from attacks of vertigo, which recurred once a week. After an interval, attacks reappeared in the spring of 1951, occurring at first three times a week. In November, they became milder, but in January, 1952, they again increased in severity. The duration varied from two minutes to one hour. The patient was compelled to lie down to avoid falling. A murmur was heard in the ear. A right-sided sympathectomy was performed in the beginning of March, 1952, with uneventful recovery. Four days after the operation the patient had an attack of vertigo lasting only one minute. When discharged from the hospital, his hearing was on the preoperative level, conversational speech 0.5-1.8 meters. The patient reappeared one year after the sympathectomy. He had had two severe attacks of vertigo two months after the operation and several slight attacks since. The right hand was still warmer than the left. During the whole following summer attacks were absent, but in the autumn they again bothered him, lasting half an hour at a time. Two years after the operation the patient informed us that he had had no more attacks. Six years after the operation he reported that he occasionally had slight attacks lasting only 1-10 minutes.

Case 3.—A man, aged 34 years, had his first attack of vertigo in the autumn, 1949. It lasted half an hour. Attacks, lasting one-half to one hour, occurred fairly often during two and one-half years. When examined, in June, 1952, he heard conversational speech with the left ear at 0.4-3 meters. Left-sided sympathectomy was performed. The first postoperative attack of vertigo occurred 13 days afterward. In the middle of August of the same year the patient reappeared. For two weeks he had had several very mild attacks of vertigo. Hearing had diminished slightly.

Case 4.—A woman, aged 30 years, had suffered from a murmur in the ear for 10 years and had had attacks of vertigo for 4 years. During that time hearing had diminished, conversational speech was heard only at 0.10-0.40 meter. Left-sided sympathectomy was performed in the beginning of April, 1954. There was a tiny rupture in the pleura, but it was sutured and the air sucked off. Recovery was uneventful. The left hand was warmer than the right after the operation. Attacks did not recur until the middle of June. She had four severe attacks, one lasting as long as six and one-half hours, after which she felt rather ill and was readmitted to the hospital. At the beginning of July she was again submitted to operation. The chorda tympani was severed, and the stapes, which was fixed to the oval window, was mobilized at the same time. Her condition remained fairly good until November, when attacks started again. Because of almost continuous vertigo, a left-sided simple fenestration was performed on Jan. 10, 1955. Two years after this she had one short attack of vertigo but has now been attack-free for three years.

Case 5.—A man, aged 37 years, had his first attacks of vertigo in August, 1953. There was an interval of 10 months, followed by more frequent attacks, almost every second day. Severe tinnitus bothered the patient. Treatment with stellate block relieved him of the attacks for one month. The attacks recurred, however, and treatment with nicotinic acid was tried. The patient had good hearing in the diseased left ear, whisper at a distance of 3-4 meters. During the following month, of November, 1954. The third thoracic ganglion there were again a couple of attacks, and left-sided thoracic sympathectomy was performed, at the end was removed, and the second ganglion was isolated with clips. Attacks were then absent till the end of February, 1955. The first, slight postoperative attack occurred in March. Objects did not seem to turn. In October of the same year, mild attacks without turning of objects recurred at intervals of about two weeks. Then followed a long period without attacks, from February to November, 1956, when they appeared again. Hearing was examined at the beginning of December, 1957. The patient heard whisper with the left ear at a distance of 3-7 meters.

Comment.—These cases are too few in number for a definite conclusion to be drawn. In two cases the attacks already reappeared 4 to 13 days after the sympathectomy, although in Case 2, for example, the higher temperature of the hand

on the side operated on was evidence of the technical success of the operation. In Case 5, however, the sympathectomy probably alleviated the attacks.

Comment

Our results show that the statement of Harrison concerning the ineffectiveness of decompression operations on the perilymphatic space does not hold good. The attacks of vertigo subsided in 78.57% of 56 cases, and another 3 patients had only a few attacks after the fenestration and were later free from attacks for several years. Thus the percentage of patients benefited rises to 83.92%. This fact does not mean that a simple fenstration is a method to be regularly applied in Ménière's disease. We know from the experience of many authors, of whom Cawthorne may be mentioned here, that destruction of the membranous labyrinth regularly cures the vertigo and giddiness although tinnitus in many cases is not affected by it.

The simple fenestration proposed by me in 1951 aimed at preservation of hearing. In my experience, unfortunately this does not inevitably result. However, from the outset, the cases have been carefully selected. First, only unilateral cases were operated on. Second, the majority already had considerably impaired hearing; in only a few cases was auditory capacity of practical value.

The operation of Dandy, dissection of the vestibular nerve in the posterior cranial fossa, ensures freedom from vertiginous attacks and giddiness and may in many cases preserve the hearing. Yet even this operation is a palliative one, and, although the danger to life has been much reduced, in the hands of modern neurosurgeons, it is understandable that the operation is used to a very limited extent. Besides, it does not attack the causative factors of the disease.

Draining of the endolymphatic sac into the cerebrospinal fluid was proposed by Portmann as early as 1926. The idea is rational, but only few have been able to report lasting and good results. We may mention, for example, Flett, in whose experiments vertigo was relieved and hearing retained in 25% of the cases, vertigo relieved but hearing gradually lost in 61%, and failures accounted for only 14%, a very good result indeed. Many authors, however, have remarked on technical difficulties, especially in avoiding hemorrhages and the tendency to closure of the "fenestra" in a later stage.

Another method of draining the endolymph was proposed by Altmann and Montreuil. The membranous horizontal canal was dissected and the endolymph allowed to drain into the perilymphatic space. This operation seems not to have been practiced widely. Therefore the outcome concerning the attacks is uncertain, but the labyrinth seems to lose its function.

Experiments on animals and observations on humans have shown that the sympathetic nerves have an influence on the vascular system in the labyrinth. On the assumption that vasoconstriction was a cause of the hydrops in the labyrinth, the blocking or cutting of the sympathetic was a natural consequence in the series of therapeutic efforts. The largest experiment has been presented by Passe. He performed blocks of the stellate ganglion and stellectomies and upper dorsal sympathectomies in which the second and third ganglia were extirpated. Both operative procedures relieved the patients completely of the attacks in 61%-62%, and 35% were in addition improved. Hearing was improved substantially in 26% and 36% and slightly in 33% and 31%. Even tinnitus was relieved or reduced in the majority of cases. Cawthorne, Lewis, and Harrison have also tried this method on smaller series of patients but have not achieved equal results. Dix examined five patients on whom Passe had performed an upper dorsal sympathectomy and was favorably impressed by the results. In my four patients the attacks persisted, being perhaps somewhat milder than preoperatively. Hearing did not show the gradual falling

which was usual after a fenestration. In one of the patients the operation was a complete failure, but a fenstration relieved the patient of the attacks. As noted, our results were not favorable enough to recommend sympathectomy as a method of choice in severe cases.

Very recently a new method devised by Krejci and Arslan has been introduced. Ultrasound is directed toward the membranous vestibular laybrinth without touching the cochlea. The attacks disappear; the hearing can be preserved, and the tinnitus is favorably influenced. It seems probable that, with an increase in the number of cases thus treated, this method will become more used and appreciated. To the present I have had no personal experience with it.

As already stated, it is very surprising that after a simple fenstration hearing should deteriorate and even be lost within a short time in more than one-fourth of the cases. It was also suggested above that in Ménière's disease there must be some special factor, not identical with aseptic labyrinthitis, which is perhaps the main cause of this deterioration. Lindsay has made a similar observation in his fenestrated patients. However, his technique differed in one detail: he left the fenestra uncovered, whereas I closed it in the usual manner with a thin cutaneous pedicled flap. After fenestration by Altmann's method hearing loss was also ascertained. In his method, as described above, the membranous canal was opened.

How is this hearing loss after fenestration to be explained? What are the changes in the organ of Corti in Ménière's disease? Hallpike and Cairns found degenerative changes in the organ of Corti in some cases; some other investigators found compression, but, since these changes have been absent in many cases, the concept is not yet clearly defined. Lindsay (1942) is of the opinion that functional disturbance in the hearing organ cannot be explained on the basis of increased endolymphatic pressure "because of the existing mechanism to preserve pressure equilibrium with the perilymph and the cerebrospinal fluid." For my part, I agree with him on one point: that it seems improbable that the pressure alone is the cause, although I do not believe in the role of the cochlear aqueduct. In an earlier anatomic study I found the aqueduct often almost obliterated in adults. This is also in accord with the clinical findings. We know that cerebrospinal meningitis rarely deafens adult patients. As our knowledge of the pathology of Ménière's disease is still deficient, it is the more difficult to answer the question concerned. We can only suggest hypothetical answers. It may be assumed that fenstration has altered the pressure conditions. Because of a relative anoxia due to already prevailing increased pressure, there exists a hypersensitivity in the hearing end-organ and perhaps, also in the nerve endings which regulate the secretion of the endolymph and of the perilymph. Fenestration upsets the balance between endolymph and perilymph, already unstable, and a discrepancy arises in production, perhaps also in resorption. This has a damaging effect on the previously weak end-organ of hearing. The consistency of the endolymph may also change. To the present time, the fluid circulation within the cochlear duct itself has not been fully explained. We do not know how the fluid in the tunnel of Corti and around the hair cells and supporting cells communicates with the main part of the endolymph in the scala media. The equilibrium may be upset here also. It is hardly necessary to add that the condition of the internal ear varies from patient to patient. From this it follows that the effect of fenestration must also differ.

How is it possible that an ordinary simple fenestration causes the vertiginous attacks to subside in the majority of cases? Do the results here presented help to clarify this question?

It may be assumed from the effect of destruction of the vestibular part of the membranous labyrinth that the site of the causative factor of the attacks must be in the vestibular end-organs. This conclusion can be drawn even more definitely from the

results of Arslan, who succeeded in destroying the vestibular sensulae of the cochlea remaining outside the sphere of the ultrasonics. However, these facts allow no conclusions as to the exact part of the end-organ where the symptoms have their origin. It may be the ampulla of the lateral canal, perhaps the other ampullae together, or the utricle. It seems less probable that the saccule would contain the focus. Yet it is all the more remarkable that the progress of the disease in the cochlea seems to become interrupted when the static end-organ is destroyed by ultrasonics.

By fenestration there will be induced an immediate change in the pressure of the perilymph. One would expect the pressure to return to its previous level after a certain time. At any rate, an influence has been set up that is directed not only toward the perilymph pressure but also probably toward the ampulla of the lateral canal and perhaps even toward the utricle. There are proofs of such an influence upon the ampulla. Of 44 fenestrated cases, 14 showed a negative caloric reaction. Of the positive 33 cases, 19 had a weaker caloric reaction. In only six the caloric nystagmus was reversible when the head was moved to the opposite position.

It was a noteworthy finding that, of the 33 fenestrated patients with a positive coloric response in the fenestrated ear, 26 were relieved of the vertiginous attacks, and, of the 7 who still suffered from attacks, 4 had only slight attacks. Schuknecht contends that a permanent healing is obtained only when caloric reaction is extinct. This does not hold good in our cases. On the other hand, we could show that a negative caloric reaction does not ensure freedom from attacks. Of the 14 patients with negative caloric reaction, 9 were free from attacks while 5 still suffered from them.

It is probable that an ordinary fenestration affects chiefly the lateral ampulla, but it is also possible that the utricle, situated in the immediate vicinity, is involved in the sphere of effect. Pursiainen and I have earlier tried to explain the irregularities in the outcome of vestibular tests due to adhesions formed between the ampulla and the new fenestra. The conditions may be more complicated however. The efferent and afferent nerve fibers of the ampullary branch of the vestibular nerve may be affected by the operation. There is evidence pointing to the existence of a secretory organ for the endolymph in the vicinity of the ampulla. Most recently the experiments of Ormerod, Dohlman, and McLay have argued in favor of such an organ. Fenestration in this region may cause a disturbance in the function of this organ. This may also help explain the change in the condition after fenestration.

This explanation is applicable to cases fenestrated in the usual way close to the ampulla but does not cover cases in which the fenestra is made further back from the ampullar region. Even in this series most of the patients were cured of attacks of vertigo. At the moment there is no definite explanation, but perhaps the changed anatomy may offer some elucidation. The vascular system of the internal ear, in the natural state quite separate from that of the middle ear, has been connected through the fenestra with the vessels of the middle ear. This alteration may affect the conditions in the perilymph and perhaps also indirectly in the endolymph.

The above considerations are hypothetical and not based on factual evidence. However, they may help to clarify the positive results of fenestration in some way. Explanation of the negative results will be more difficult.

Summary

A series of surgically treated patients with unilateral Ménière's disease is presented. Of these patients, 56 were submitted to fenestration of the lateral semicircular canal, 47 to an ordinary fenestration, and 9 to a fenestration on the vertex of the canal. In addition, primary destruction of the membranous labyrinth was performed on three patients and upper

dorsal sympathectomy, according to Passe, on five.

In 78.57% of the cases attacks subsided. If 5 patients observed for less than one year are excluded, 41 of 51 got rid of attacks, the percentage being 80.3%. The behavior of the response to caloric and rotation stimulation in the ear operated on is reported. Tinnitus was not relieved worth mentioning. In the majority of ears operated on hearing deteriorated sooner or later. The cases in which the fenestra was made behind the ampullar region are treated separately. The postfenestration working capacity of the patients is discussed.

The cases in which the membranous labyrinth was primarily and secondarily destroyed are described. Five upper dorsal sympathectomies are reported.

An attempt is made to explain the subsidence of the vertiginous attacks after fenestration and the effect of fenestration on hearing. It seems that a disturbance of the balance of the endolymph and perilymph production is the cause of the change in the behavior of the internal ear. It is probable that the new fenestra affects the ampullar end-organ in some way.

Conclusions

A conventional fenestration of the affected ear in Ménière's disease relieves about 80% of patients of vertiginous attacks. In the majority of cases with a positive postoperative fistula test, freedom from attacks was established. In most cases with a negative fistula test, attacks also subsided.

In both groups, with positive and negative postoperative caloric reaction, the majority of patients were free from vertiginous attacks. Thus, extinct caloric reaction after fenestration is not a requisite for freedom from attacks. No correlation was found between postoperative sensitivity to rotatory stimulation and occurrence of postoperative attacks of vertigo. Freedom from attacks affects the working capacity favorably.

Although the site of the surgical activity in fenestration is far from the cochlea, hearing will not be preserved. On the contrary, the operation seems in many cases to hasten development of further hearing loss. The latter cannot be due to possible aseptic labyrinthitis alone. There must be a special factor in the internal ear, peculiar to Ménière's disease.

In cases with a negative result, secondary destruction of the membranous labyrinth after an ordinary fenestration leads to freedom from attacks.

In my experience, upper dorsal sympathectomy does not ensure freedom from attacks; only partial alleviation may result.

Fredrikinkatu 20A.

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Manipulation of the Labyrinth Windows and Their Surroundings in Tympanoplasty

Clinical Observations

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Introduction

Tympanoplasty consists of a series of operative procedures, which not only eliminate chronic infections of the middle ear completely but also improve hearing. The purposes of tympanoplastic procedures are the elimination of inflammatory processes and postinflammatory changes in the middle ear in order to diminish ear discharge and to obtain dry ear and the reconstruction of the damaged sound-conduction mechanism in order to facilitate sound conduction to the inner ear and restore the hearing.

In our experience there are cases where tympanoplasty failed to eliminate the inflammatory processes or to restore hearing in chronic otitis media. I have, therefore, clinically observed these cases of failure in order to clarify manipulation of the labyrinth windows and their surroundings and assure accomplishment of both aims of tympanoplasty.

Submitted for publication Dec. 16, 1958.

From the Department of Oto-rhino-laryngology, Nagoya University School of Medicine (Director: Prof. S. Goto).

Material

I examined 242 tympanoplasty cases operated on by us for chronic middle ear suppuration from October, 1957, to November, 1958.

The cases included are chronic otitis media (193), cases following radical mastoidectomy (33), and those following tympanoplasty (16), totaling 242.

The cases following radical mastoidectomy and tympanoplasty who complained of ear discharge or who were hard of hearing were operated on.

	Cases, No.	Ear Discharge	Hard of Hearing
After radical mastoidectomy	33	30	3
After tympanoplasty	16	10	6

GROUP 1.—First arises the question of determining the causes of failure in the 16 cases where ear discharge continued even after operation and the 8 cases where hearing was not improved after

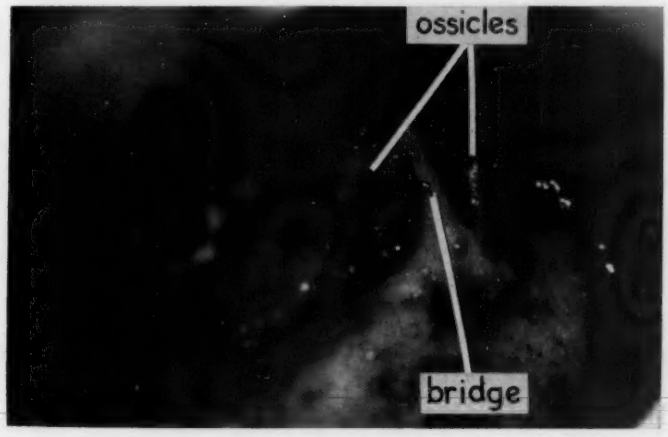


Fig. 1.—Bridge and ossicular chain.

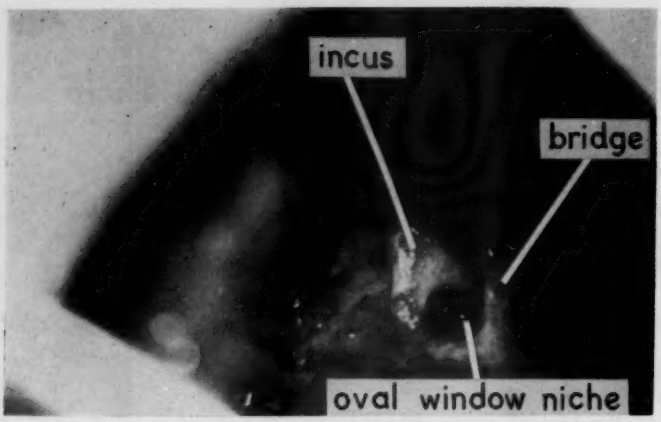


Fig. 2.—Oval window and niche.

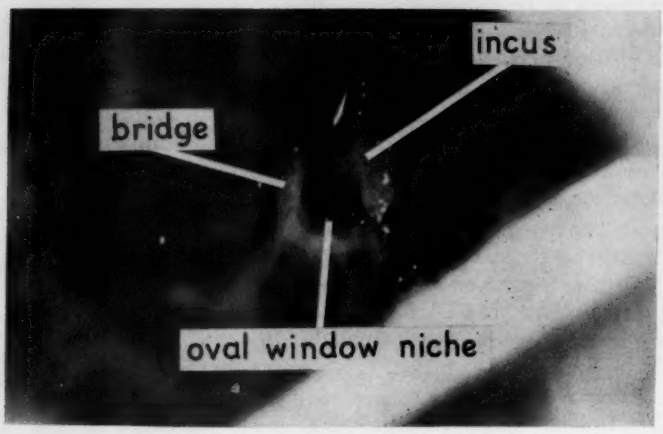


Fig. 3.—Oval window niche.

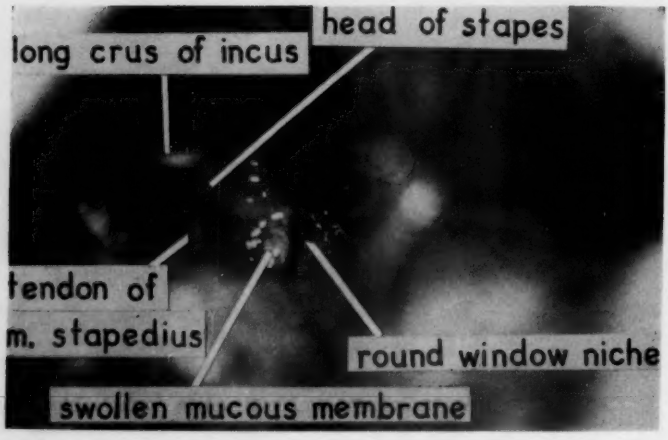


Fig. 4.—Swollen mucous membrane covering round window niche.

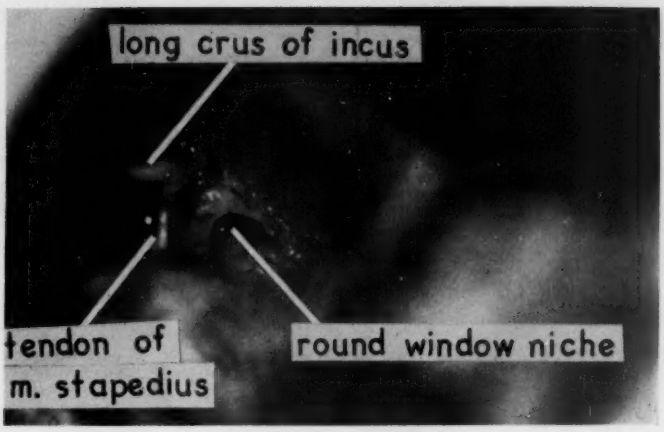


Fig. 5. — Round window niche after removal of covering mucous membrane, as shown in Figure 4.

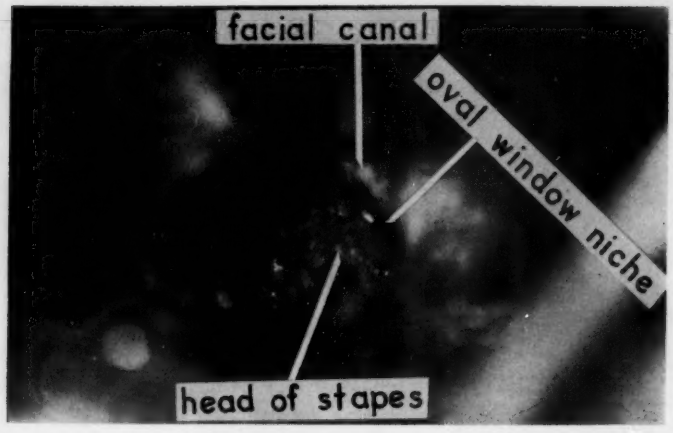


Fig. 6. — Stapes and oval window niche.

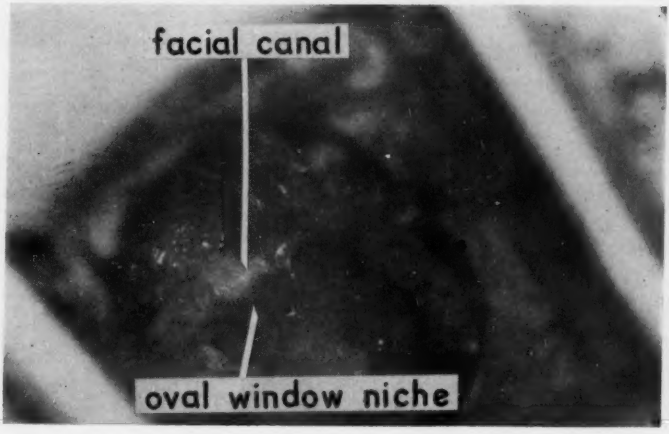


Fig. 7.—Oval window niche without stapes.

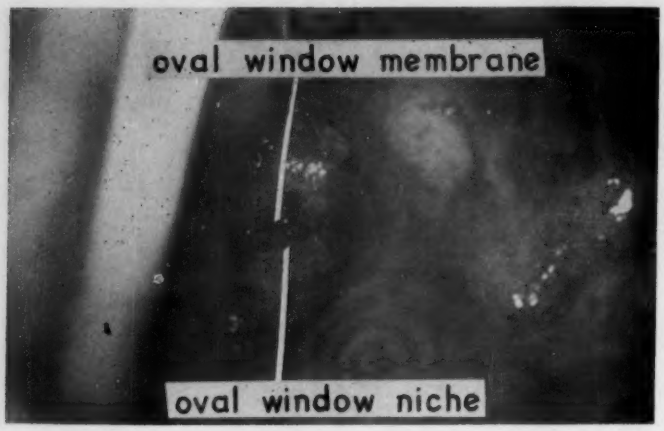


Fig. 8.—Oval window membrane.

Fig. 9.—Stapes with swollen mucous membrane.

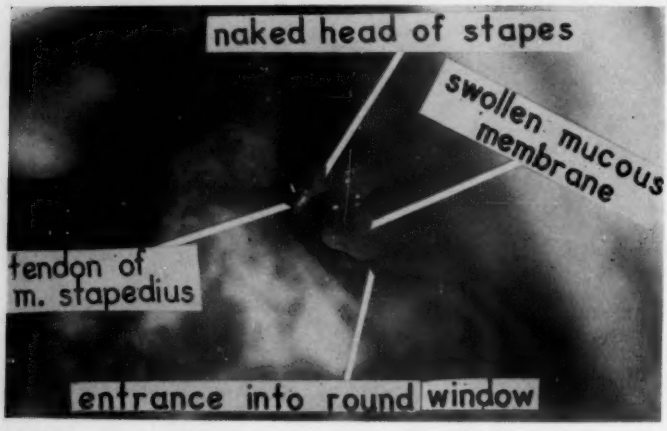


Fig. 10.—Naked head of stapes and round window niche covered with swollen mucous membrane.

tympanoplasty out of the 193 cases of chronic otitis media.

GROUP 2.—Second arises the discussion of the 10 cases in which the aims of tympanoplasty were attained for only a certain period but in which ear discharge was again a complaint recently, and also of the 6 cases where the aims of tympanoplasty were accomplished for a certain period but which presented a recent complaint of being hard of hearing.

I performed reoperation in these 40 cases of failure of tympanoplasty and observed the pathology in the middle ear at operation.

Results

GROUP 1.—A. Revision operation for 16 cases of ear discharge after tympanoplasty for chronic otitis media.

Of eight cases with preserved ossicular chain, swelling of the mucous membrane was seen around the ossicular chain in four and granulomatous swelling of the mucous membrane around the stapes in four. In eight cases with only the stapes preserved or with no stapes, there were granulations in the labyrinth window niches and the hypotympanum.

B. Reoperation on eight not discharging but hard of hearing ears after tympanoplasty for chronic otitis media.

These consisted of three cases with dry perforations of newly formed tympanic membrane, three with discontinuity between the head of the stapes and the new drum membrane in cases with only the stapes, and two with no direct covering of the epidermis extending from the drum membrane on the oval window in cases with no stapes.

GROUP 2.—A. Reoperation for 10 discharging ears, which had been exempt from both infection and difficulty in hearing for some period after operation.

In three ears with preserved ossicular chain there were found swellings of the mucous membrane around the ossicular chain, especially around the stapes. In five cases with only the stapes there were found granulomatous mucous membrane or a small amount of cholesteatoma at the stapes region, in the tympanic sinus, or in the tympanic cavity. There were found granulations in the oval window niche and its surroundings in two with no stapes.

B. Revision operation for six hard of hearing ears, which had been freed from both infection and difficulty in hearing for some time after operation.

In five cases there were found dry perforations of the tympanic membrane, and in one ear mobility of the stapes was found to be restricted by connective tissue formation.

Next, tympanoplastic surgery was performed in cases with ear discharge or which were hard of hearing after foregoing radical mastoidectomy or conservative radical mastoidectomy.

Of 30 cases in which radical mastoidectomy or conservative radical mastoidectomy had been performed before but aural discharge continued or sometimes reappeared, granulations were found at the oval window niche in 2 cases; in 11 cases granulations were observed at the labyrinth window niches and the promontory and/or the hypotympanum; in 5 cases there were granulations and pseudocholesteatoma at the stapes; granulations at the stapes region and infection in the perilabyrinthine cells were found in 8 cases, while in 2 cases granulations were found in the tympanum and the tubal region, and there were 2 cases of bony infection of the promontory.

In three cases in which radical mastoidectomy had been performed before and aural discharge was not observed but difficulty in hearing was complained of, there was in one case of ankylosis of the stapes by fibrosis, bony fixation of the footplate of the stapes, and direct covering of the epidermis, spreading from the promontory over or into the round window niche, without formation of a small tympanum in which the Eustachian tube and the round window area should be involved.

Lastly, I collected cases of failure after tympanoplasty in order to determine whether operative success differs from the original conditions of the tympanic cavity, i. e.,

chronic otitis media, after radical mastoidectomy, or after tympanoplasty.

1. Failure cases after tympanoplasty performed in 193 cases of chronic otitis media included 16 with continued ear discharge and 8 which continued to be hard of hearing, as was already mentioned above, i. e., failure of tympanoplasty in chronic otitis media was 12.4%.

2. Failure cases of tympanoplasty performed in 33 cases of radical mastoidectomy included 8 with continued ear discharge and 3 which were hard of hearing, i. e., failure of tympanoplasty in radical mastoidectomy was 33.3%.

3. Failure cases of tympanoplasty performed in 16 cases which had been freed from both infection and difficulty in hearing for some period after first operation but who recently began to complain of ear discharge or difficulty in hearing, included 2 with continued discharge and 2 which were hard of hearing after a second tympanoplasty, i. e., failure cases of tympanoplasty for those complaining of ear discharge or difficulty in hearing after a certain period following the first operation was 25%.

4. Failure cases of tympanoplasty performed in 24 cases after a first failure of tympanoplasty for chronic otitis media and operated on a second time very soon included 3 with continued ear discharge and 1 with continued difficulty in hearing after the second tympanoplasty, i. e., tympanoplasty failure in cases with continued complaints after a first tympanoplasty for chronic otitis media was 16.7%.

Comment

From the above-mentioned data we can say that in cases of preserved ossicular chain, there is fear that the irreversible pathologic mucous membrane around the ossicular chain will remain so even if a tympanoplasty is performed. Also, the irreversible pathologic mucous membrane at the labyrinth window niches, tympanic sulcus, and hypotympanum may remain. This irreversible pathology must be assessed and completely eliminated during operation. One can assess irreversible pathology as osteitis, granulations, or granulomatous or hypertrophic mucous membrane with an operative microscope.

For the hard of hearing cases after tympanoplasty the following precautions should be taken: To prevent a perforation of the newly formed tympanic membrane it is necessary to maintain nutrition of the drum membrane and the patient must be careful in blowing the nose; to prevent disturbance of the sound conduction from the tympanic membrane to the inner ear after tympanoplasty it is necessary and important to remove completely the mucous membrane covering the head of the stapes, to bring into intimate contact the head of the stapes with the drum membrane in those with only the stapes preserved, and to expose the oval window and to cover this window membrane with the epidermis from the drum membrane in cases with no stapes. To maintain nutrition of the free skin graft forming a new drum membrane, suitable bedding must be prepared in a fashion to nourish the graft and eventually provide a blood supply; to do this it might be necessary, according to circumstances, to strip the outer cuticle of the drum head and the skin of the adjacent auditory canal.

There were cases with some pathologic mucous membrane after tympanoplasty which might be healed, as well as those with some pathologic mucous membrane after tympanoplasty in whom discharge might reappear. This presents the difficulty of recognition of irreversible pathology, and to facilitate the recognition of irreversible pathology it is necessary to use more than tenfold magnifying glass during operation.

A perforation of the drum membrane may be seen after a certain period following tympanoplasty. This resulted from neglect in observation and manipulation of the healing tendency of the newly formed drum membrane, i. e., there may be deceptive healing due to crust covering the drum membrane. To prevent a perforation one must clear up the squamous epidermis of

the graft 7 or 10 days after operation. To prevent secondary ankylosis of the stapes it is necessary to retain a small amount of the mucous membrane at the footplate of the stapes and if possible at the foot of the stapes in order to regenerate rapidly the mucous membrane around the stapes.

In most cases with radical mastoidectomy there remained infection of the pneumatic cells and/or pathology of the stapes region.

It is necessary to ensure complete success of tympanoplastic surgery at the first operation. If the first operation should not succeed, a second operation should be performed as soon as possible.

There is a close relation between the pathology of the stapes region and postoperative inflammation. It is extremely important for improving postoperative hearing that in cases with only the stapes or with no stapes the stapes or the oval window should be in close contact with the newly formed drum membrane. Under these circumstances we can determine the method of manipulating the stapes region in tympanoplastic procedures. That is, to eliminate infection of the middle ear the irreversible pathology must be completely removed. This is easy to say but not always easy to perform, i. e., even if manipulation looked complete with the naked eye or with a severalfold magnifying glass, there may be observed a remaining pathology with more than a tenfold magnifying glass under continuous irrigation and suction, and the remaining pathology at the stapes region and its surroundings should be carefully eliminated by specially manufactured small instruments.

In cases of intact ossicular chain tympanoplastic procedure must be manipulated to discover the oval and round window niches (Figs. 1-5).

In cases of only the stapes the pathology around the stapes must be removed to expose the stapes and to observe the oval window niche (Figs. 6 and 10).

In cases of no stapes the pathology in the oval window niche must be removed to expose the oval window (Figs. 7 and 8).

To improve hearing after tympanoplasty the sound-conduction mechanism must be reestablished. The skin graft over the tympanum should be carefully manipulated. Special care must be taken in cases of only the stapes and of no stapes. In cases of only the stapes the mucous membrane covering the head of the stapes must be removed to expose the bony surface (Figs. 9 and 10) and the new drum membrane must be brought into contact with the naked head of the stapes. In cases of no stapes the pathology in the oval window niche must be removed to expose the oval window membrane, and the epidermis spreading from the new drum membrane must be brought into contact with the oval window membrane.

Summary

To obtain complete healing and restoration of hearing after tympanoplasty for chronic otitis media, I examined 242 tympanoplastic cases operated on from October, 1957, to November, 1958. Of the many problems which present themselves in the restorative surgery for middle ear disease, I could pick out only a few, and these were merely of an operative technical nature.

In tympanoplasty we must discover the oval and round window niches, and the irreversible pathology herein must be removed completely under operative microscope to obtain a complete healing.

To improve hearing after tympanoplasty, sound-conduction mechanism must be reestablished. The skin grafting over the tympanum should be carefully manipulated, and special attention must be paid to cases with only the stapes and with no stapes. In cases with only the stapes the naked head of the stapes must be brought into contact with the skin graft to build a shallow tympanum. In cases with no stapes the oval window membrane must be brought into contact with the epidermis spreading from the new drum membrane to build a small tympanum.

Department of Otolaryngology, Nagoya University.

Vital Staining of Cervical Lymphatic Nodes by Means of Direct Sky Blue

Author's Experiences in Radical Neck Dissection

RYUICHI KUBO, M.D., Kagoshima, Japan

The purpose of this paper is to describe my experiences in the staining of cervical lymphatic nodes in cases of malignant tumors of the upper respiratory tracts with direct sky blue to facilitate the radical neck dissection.

It is a well-known fact that malignant tumors of the upper respiratory tracts, including the oral cavity, form metastasis in cervical lymphatic nodes. In spite of this fact, it cannot be denied that the treatment of cervical lymphatic nodes in the surgery of malignant tumors in our field has been rather conservative. Almost all surgeons who are engaged in treating malignant tumors of the upper respiratory tracts would have seen, if they had neglected unpalpable cervical lymphatic nodes or if they had extirpated only a few palpable ones, that metastasis following the operation was often fatal for patients. This is the reason why radical neck dissection has been advocated. Indeed, we can often find cancer cells histologically in clinically unpalpable lymphatic nodes which are no larger than a pea. These small lymphatic nodes are often apt to be disregarded in radical neck dissection. If these small lymphatic nodes, including those which have already swollen, are selectively stained, they could be distinctive objectives in radical neck dissection, and, moreover, the visible lymphatic nodes could be more thoroughly extirpated. For the purpose of vital staining of cervical lympatic nodes I used direct sky blue (made by Tomoda Pharmaceutical Co., Japan), and I want to describe my experiences.

Submitted for publication Dec. 9, 1958.

Professor of Otolaryngology and Director of the Ear, Nose, and Throat Clinic, Faculty of Medicine, Kagoshima University.

Description of Direct Sky Blue

Direct sky blue is a dark blue crystal soluble in water, and it does not become discolored by acids and alkalis. The formula for direct sky blue is given in Figure 1.

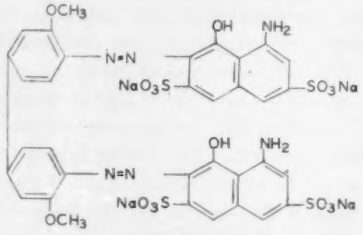


Fig. 1.—Direct sky blue.

McMaster and Parsons were the first who stained lymphatic nodes vitally with pontamine sky blue, in animal experiments in 1939. Later, in 1950, Weinberg and Greaney applied it in surgery of lung and gastric cancer. The dyestuff has been further applied in surgery of mammary, gastric, and rectal cancer, in Japan, and its superiority has been highly esteemed, but I have never seen such reports in the field of otolaryngology.

In clinical uses the dyestuff must be in conformity with the following conditions:

1. It must be easily soluble in water.
2. The tone of the stained color must be distinct.
3. It must have special affinity with lymphatic tissues.
4. Its toxicity must be at the minimum.

Direct sky blue is the 3.5% aqueous solution of the crystal. Concerning the toxicity, the LD_{50} is 378.5 mg. per kilogram. Therefore, if a patient weighs 50 kg. and if 5 cc. of the solution is injected, the dosage of the direct sky blue is only 3.5 mg. per kilogram, and accordingly its toxicity is beside the question. Moreover, the tissue into which the solution is injected is extirpated, and

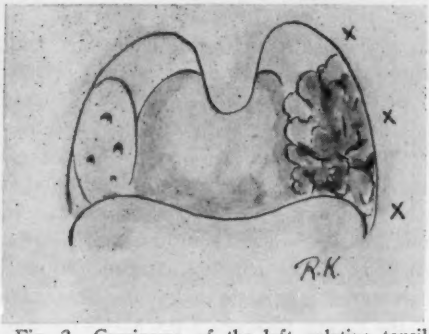


Fig. 2.—Carcinoma of the left palatine tonsil. The *x* marks show the points into which direct sky blue is injected.

the absorbed dyestuff can be readily excreted in the urine without any damage to other organs.

Experiences in Use of Direct Sky Blue

I have hitherto applied direct sky blue in cases of malignant tumors of the palatine tonsil and the tongue.

1. *Carcinoma of the Palatine Tonsil.*—The points of injection are illustrated in Figure 2. In the first case, I injected 5 cc. of the solution at three points.

As the patient feels pain when the solution is injected into the peritonsillar tissue, it is better to infiltrate anesthetic at the same points previously. In this case, the radical neck dissection was carried out five hours after the injection. The skin of the face and chest turned faintly blue, but in the field of operation of the neck the superior and inferior cervical profound lymphatic nodes of different sizes and the lymphatic vessels connecting each other were distinctly stained with blue. Severing the sternocleidomastoid muscle at its sternoclavicular origin, I could find small lymphatic nodes distinctly stained with blue in the alveolar tissue of the supraclavicular fossa. If the operator removes all of the colored lymphatic nodes, together with the sternocleidomastoid muscle and the alveolar tissues en bloc, the radical neck dissection can be performed far more completely.

In the second case, I injected 3 cc. of direct sky blue into the peritonsillar tissue, just as in the first case, and the radical neck dissection was carried out one hour after the injection. In this case, staining of the cervical lymphatic nodes and lymphatic vessels was very satisfactory, just as in the first case. From these experiences I have come to perform radical neck dissection about one hour after the injection of 3 cc. of the solution.

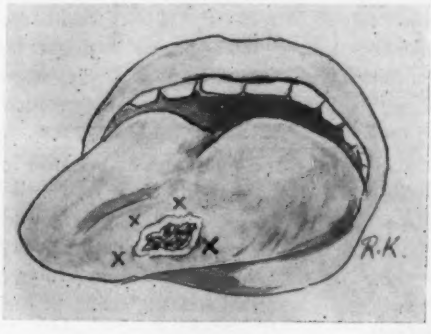
In the second case, I saw a few lymphatic nodes swollen as large as the egg of a dove, and they were also stained distinctly. Metastasis of cancer cells in these lymphatic nodes was later confirmed by histological examination.

I do not want to describe here the method of operation for extirpation of the primary tumor of the palatine tonsil in detail. However, it might be emphasized that the extirpation of the primary tumors, although the surrounding tissues turned blue, is not disturbed by staining.

Patients with tonsillar malignant tumors were given injections with 3-5 cc. of the solution (some of them were given 4 cc. of the solution in each side, bilaterally 8 cc.). Accordingly, the skin of the face, neck, and body turned faintly blue, and the tongue, soft palate, posterior wall of the pharynx, and arytenoid regions also turned darker blue. But the faintly blue color of the skin disappeared in less than 22 days, the dyestuff being excreted in the urine.

2. *Carcinoma of the Tongue.*—In these cases it was enough to inject 3 cc. of the solution around the tumor to stain cervical

Fig. 3.—Carcinomatous ulcer at the left margin of the tongue. The *x* marks show the points into which the solution is injected.



as well as submental and submandibular lymphatic nodes (Fig. 3). It was also necessary to infiltrate anesthetic into the tissue around the tumor prior to the injection of direct sky blue. In cases of the cancer of the tongue, it is especially necessary to extirpate all the submandibular and submental lymphatic nodes, together with the submandibular and sublingual salivary glands.

In every case of cancer of the tongue, if 3 cc. of the solution is injected around the tumor, the whole tongue and the mucous membrane of the floor of the mouth turned blue after one hour but the ulcer remained unstained, like an island.

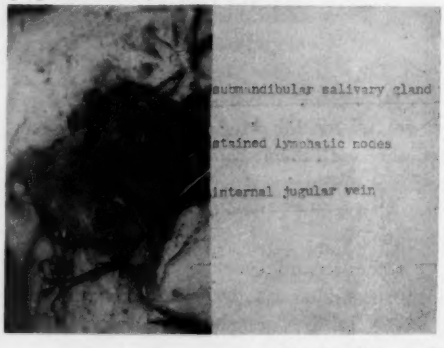
When the cancer has originated at the anterior and middle one-third of the lingual margin, the submandibular, submental, and superior profound cervical lymphatic nodes began to stain four minutes after the injection of direct sky blue. The submandibular salivary gland also turns blue, to say nothing of the sublingual salivary gland. But if the cancer is located at the posterior one-third of the tongue, the dyestuff injected begins to stain superior cervical lymphatic nodes and those on the scalenus muscle only. In this case, submandibular, submental lymphatic nodes and submandibular salivary gland do not become stained.

In each case the staining process along lymphatic vessels proceeds to superior and inferior cervical lymphatic nodes, and the staining stops about 60 minutes later. These observations were done in the neck wound from which the sternocleidomastoid muscle was experimentally removed prior to the injection of direct sky blue around the primary tumor. Accordingly, it might be concluded that the radical neck dissection should be begun one hour after the injection of the solution around the primary tumor.

In regard to the coloration of the skin in these cases, the skin of the face, of course, turns faintly blue and the whole tongue, except the ulcer, also turns dark blue, to say nothing of the mucous membrane of the floor of the mouth—and the urine of the next day. But the faintly blue color of the skin disappears seven days later, and that of the urine does so after six days. However, the color of the mucous membrane and the remaining part of the tongue can remain 50 days later, though it becomes extremely faint. If direct sky blue is injected after the origin of the sternocleidomastoid muscle is severed and radical neck dissection is carried out immediately after the complete staining of lymphatic nodes, the blue color of the tongue disappears six days after and that of the skin seven days after, and, moreover, the urine is not distinctly blue. In other words, the shorter the time between injection of direct sky blue and the radical neck dissection, the sooner the blue color of the skin, mucous membrane, and the remaining part of the tongue disappears.

When the cancer is located near the apex of the tongue and direct sky blue is injected around it, the staining process of lymphatic nodes proceeds as described above. But in these cases metastasis of lymphatic nodes of the opposite side should be considered. In a recent case, in which the cancer was located at the right lingual margin near the apex, the radical neck dissection and the extirpation of the tumor were carried out under the injection of direct sky blue. Thirty-five days later the prophylactic radical neck dissection was carried out at the opposite side, and I could certify that the submental, submandibular, superior, and

Fig. 4.—Stained lymphatic nodes in the field of radical neck dissection. (The sternocleidomastoid muscle is already removed to show the stained lymphatic nodes.)



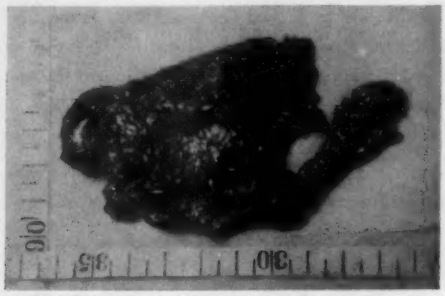


Fig. 5.—Stained lymphatic nodes in the dissected tissues of the neck.

inferior cervical profound lymphatic nodes, together with the submandibular salivary gland, had been distinctly stained. From this fact, I found that the staining of lymphatic nodes and salivary gland of the opposite side remained even 35 days later.

Comment

In modern surgery of malignant tumors of the upper respiratory tracts it is routine to carry out therapeutic or prophylactic radical neck dissection. No matter how completely primary tumors are extirpated, permanent cure of the malignant tumors in such regions cannot be expected without radical neck dissection.

Radical neck dissection is the procedure in which, if any metastasis of cervical, submandibular, and submental lymphatic nodes is clinically palpable or is doubtful, the operator removes the sternocleidomastoid muscle, not to mention metastatic lymphatic nodes, all visible lymphatic nodes, and the internal jugular vein, if necessary, together with alveolar tissues en bloc.

If regional lymphatic nodes are selectively and distinctly stained vitally, it will help the surgeon to carry out radical neck dissection far more completely and easily. Especially in cases of prophylactic radical neck dissection this method of vital staining of lymphatic nodes has an important meaning.

Ideally speaking, dyestuff that could selectively stain only metastatic areas is desirable. Of course, direct sky blue is far from this requirement, but, if there are no disturbances of lymphatic streams, it can stain almost all regional lymphatic vessels and nodes, whether they are metastatic or not.

According to Kajitani et al. (Japan), if there are many metastatic lymphatic nodes or if there is edema at the site of the primary tumor, the lymphatic stream can stagnate there or the stream can take irregular courses. In one of my cases, when direct sky blue was injected directly into the tumor, cervical lymphatic nodes did not turn blue. Moreover, in cases of cancer of the tongue, carcinomatous ulcers always remain unstained, as if they were islands. Therefore, the staining can be disturbed when the lymphatic streams stagnate by infiltration of cancer cells.

This vital staining of cervical lymphatic nodes can be applied in cases of malignant tumors of the maxilla or the larynx, but I have had no experience worthy of description here.

If quick discoloration of the skin and the mucous membrane is desired, I would recommend the intravenous injection of PVP-Macrose, a polyvinyl pyrrolidone preparation, by which the remaining dyestuff is rapidly excreted into the urine.

Finally, I wish to add that there is no disturbance for histological examination of stained lymphatic nodes and tissues.

Summary

In order to carry out radical neck dissection in cases of malignant tumors of the upper restiratory tracts more completely, I applied the injection of direct sky blue around the primary tumor and succeeded in staining cervical, submental, and submandibular lymphatic nodes vitally and distinctly. Although there have been some defects in my experiences and even though the method is not an ideal one, it will help surgeons who are engaged in operative treatment of malignant tumors in the field of otolaryngology.

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Primary Adenoidcystic Carcinoma of the Esophagus

Report of One Case and Review of the Literature

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The actual incidence of primary glandular carcinomas of the esophagus is difficult to ascertain. In the literature adenocarcinomas are often considered to represent about 10% of esophageal carcinomas. In everyday practice at the Dr. I. González Martínez Oncologic Hospital and upon reviewing other large series of esophageal carcinomas it becomes apparent that true authenticated primary esophageal adenocarcinomas are very rare. Most of the glandular tumors of the esophagus are located at the cardia, and one may assume that all in that location are derived from gastric glands. It is a well-known fact that many of these gastric glands may be encountered beneath the lowermost portions of the esophageal mucosa. In erosive esophagitis the gastric mucosa may grow up, covering the defect. Hamperl [9] has suggested that the latter healing mechanism might be a source of adenocarcinoma of the lower esophagus. These cases, if they occur, would actually be gastric carcinomas.

McPeak and Warren,[14] in a review of 65 cardioesophageal carcinomas, suggested that 4 adenoacanthomas and 2 papillary adenocarcinomas may have originated from esophageal glands. This cannot be proved. Most authors consider glandular tumors at the cardioesophageal junction gastric carcinomas with extension to the esophagus.[1,3,5,15]

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Primary, pure glandular carcinomas arising in other parts of the esophagus are extremely rare. Stout [15] considers that most of the reports of adenocarcinomas of the esophagus are so few and so unsatisfactory that it might be questioned if the entity really exists. Ackerman and Del Regato [1] also very much doubt their existence.

In 1900, Hewlett [10] reviewed the literature and encountered four cases of adenocarcinoma in the upper two-thirds of the esophagus and two of the lower third, separated from the stomach by normal esophageal mucosa. Since that time sporadic reports of esophageal adenocarcinomas have appeared in the literature.

Feldman [6] reported an adenocarcinoma arising in an esophageal polyp, on the basis of a biopsy alone. Stout [15] believes that the information furnished in this case is so scanty that the case cannot be accepted as proved. We are of the same opinion.

Carrie [4] described an adenocarcinoma of the upper third of the esophagus arising from ectopic gastric mucosa. The case of Goldman [7] was located in the junction of the middle and lower thirds but was limited to the submucosa.

Thomas and Hay [16] reported an adenocarcinoma of the lower third, located 5 cm. from the cardia, but the adjacent esophagus had glandular metaplasia of the mucosa. This could have been the result of a healing esophagitis or a congenitally short esophagus with a tubular supradiaphragmatic stomach.[15] It is of interest to note that in none of the above mentioned cases were metastases reported.

The presence of glands in the esophagus is certain, and, if they can produce cysts,

they could also be the seat of neoplastic changes, giving origin to glandular tumors.

The serous-mucus glands of the esophagus theoretically could produce tumors resembling those seen in salivary glands. Ahlbom,[2] in a large series of adenoidcystic carcinomas, did not find a single one arising in the esophagus. McPeak and Arons [13] reported a tumor located 2 cm. above the cardia which they considered to be an adenoacanthoma and suggested the possibility that it might represent a malignant mixed tumor. A similar tumor was reported by Kinoshita.[12] Gregg and Stamler [8] reported a tumor growing as a polypoid mass with areas of fibrosarcoma, adenocarcinoma with squamous metaplasia, pure epidermoid carcinoma, and some areas resembling adenoidcystic basal-cell carcinoma. In the addendum of their article they reported another case of a tumor located in the midesophagus and showing areas of adenoidcystic carcinoma with metastases to lymph nodes.

Jackson [11] encountered 2 tumors with basal-cell characteristics and 2 mixed tumors among 671 carcinomas of the esophagus.

An adenoidcystic carcinoma of the lower end of the esophagus in a 55-year-old man is illustrated in the "Color Atlas of Pathology," of the U. S. Naval Medical School.[5] He died of inanition.

The purpose of this communication is to present a pure adenoidcystic carcinoma of the midesophagus. This represents the fourth case in the literature, as far as we know, in which areas of cylindroma are encountered in an esophageal cancer and the second case of a pure adenoidcystic carcinoma of the esophagus.

Report of Case

A 51-year-old man was seen for the first time at the Dr. I. González Martínez Oncologic Hospital on May 28, 1955, with a history of dysphagia for six months to semisolids and solids. During this time he had lost 23 lb. in weight.

Physical examination was completely noncontributory. There were no palpable cervical or supraclavicular lymph nodes. No abnormalities were encountered on pharyngolaryngoscopic examination. X-ray studies after barium swallow disclosed a stenosing ulcerated lesion of the middle esophagus measuring on the x-ray films 4 cm. in length (Fig. 1). The lumen at the point of obstruction was 1 cm. in diameter.

Fig. 1.—Radiograph after barium swallow, disclosed a stenosing ulcerated lesion in the middle third of the esophagus.

Esophagoscopy showed a lesion at a distance of 31 cm. from the upper incisor teeth. A first biopsy specimen was reported as showing acute and chronic inflammation; but a second specimen provided histologic proof of the presence of the tumor.

A thoracotomy and esophagectomy were performed on July 29, 1955. The tumor involved the middle third of the esophagus, extending about 5 cm. in its long axis. The tumor was adherent posteriorly and to the right pleura. There were several small nodes felt in the periesophageal tis-

Fig. 2.—Resected portion of the esophagus, representing the middle third and mostly occupied by the tumor. The separate portion of tissue was shaved from the periesophageal extension to show it in the photograph.

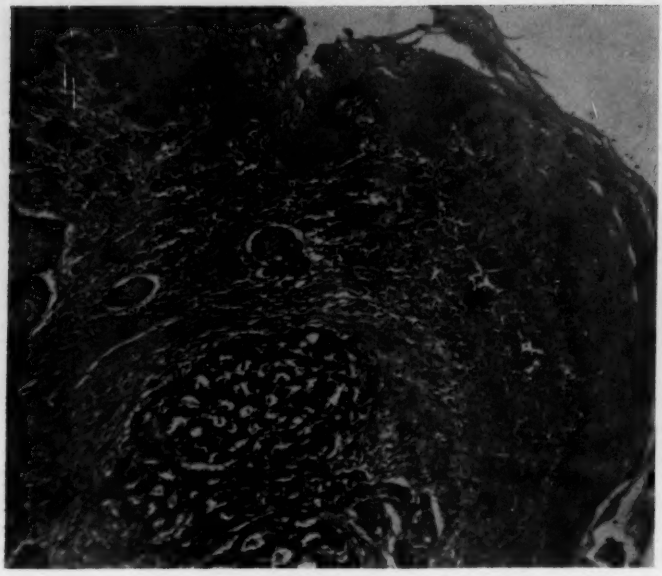

Fig. 3.—Areas of cylindroma beneath the intact esophageal mucosa. Hematoxylin and eosin; reduced approximately 15% from mag. × 80.

sues as well as along the left gastric vessels. A supra-aortic, terminolateral esophagogastrostomy was done. The patient tolerated the procedure very well and had a benign postoperative course complicated only by a temporary pleural effusion on the right, which absorbed spontaneously.

Pathologic Findings.—Gross Description: The portion of resected esophagus measured 13.5 cm. It presented an ulcerated infiltrating neoplasm measuring 7 cm. and completely encircling the esophagus (Fig. 2). In the most peripheral portions of the longitudinal axis the tumor was still covered by slightly thickened esophageal mucosa. On section, the tumor infiltrated deeply into the muscular coats and out into the periesophageal

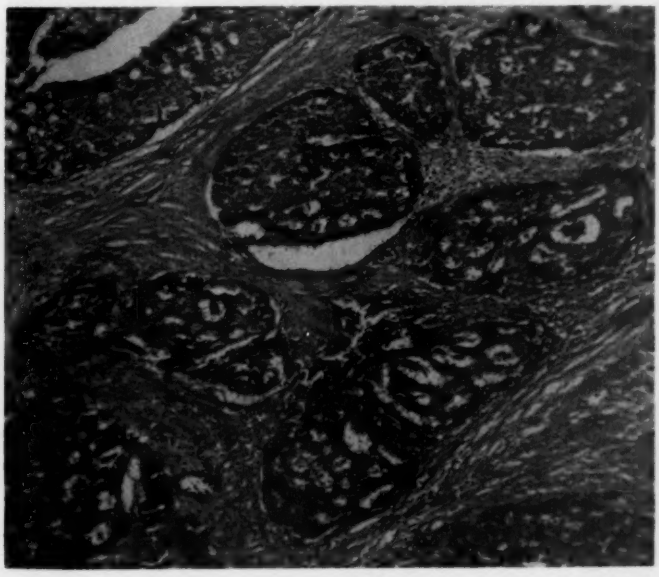

Fig. 4.—Sections from the bulk of the tumor, exhibiting the classical histologic features of cylindroma. Hematoxylin and eosin; reduced approximately 15% from mag. × 80.

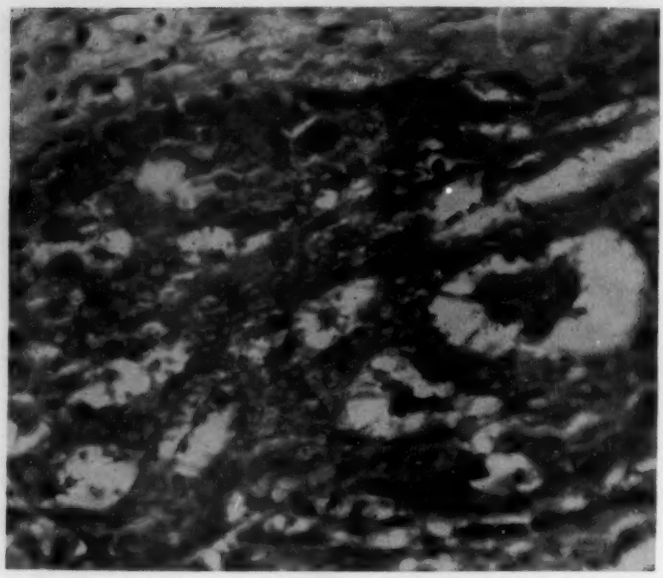

Fig. 5.—Higher magnification of the tumor. Hematoxylin and eosin; reduced approximately 15% from mag. × 360.

tissues. The gross appearance was not unlike the usual variety of epidermoid carcinoma.

Microscopic Description: Most of the surface was ulcerated and secondarily infected. Where the mucosa was intact tumor was seen beneath it (Fig. 3). The latter showed the classical histological features of an adenoidcystic carcinoma, so-called cylindroma of salivary glands. This was especially so in the deep portions (Figs. 4 and 5) of the tumor which were not altered by the ulceration, necrosis, and inflammation. Tumor was present in the perineural lymphatic spaces in the periesophageal tissues. The distal and proximal margins of the resection were free of tumor.

Clinical Course.—Six months after the esophagectomy he was very much improved, swallowing all foods without any difficulty, and had gained weight. In the seventh postoperative month he

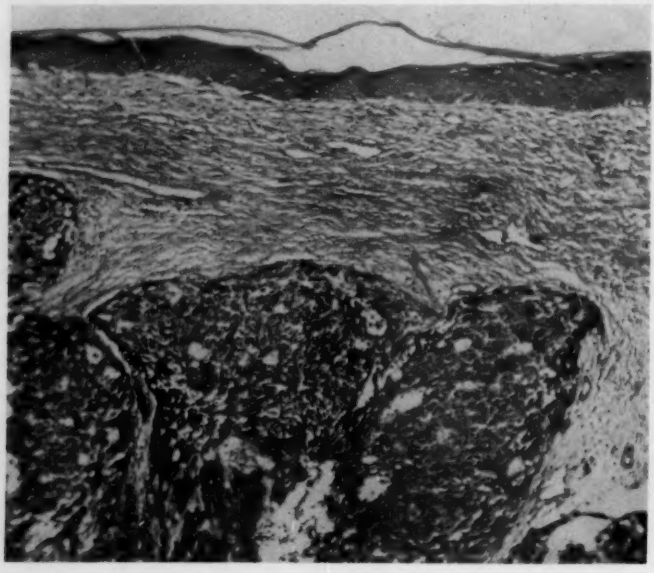

Fig. 6.—Metastases of the tumor to the subcutaneous tissues of the upper lip. Hematoxylin and eosin; reduced approximately 15% from mag. × 80.

complained of several subcutaneous nodules which had appeared in the upper lip, palm of the left hand, right index finger, and right second and left fourth toes. Several of these lesions were removed for diagnostic purposes, disclosing a histologic pattern similar to that of the primary esophageal lesion Fig. 6).

The patient died at his home in December, 1956, two years after the onset of his disease and 18 months after esophagectomy. An autopsy was not obtained.

Comment

From a review of the literature on primary glandular carcinomas of the esophagus, it is obvious that, when carefully analyzed, most cases are open to criticism and the great majority are gastric carcinomas with esophageal extension. It is also true that, although the existence of this entity is not fully accepted by some authorities, we most definitely recognize it. It seems from the reports available and from our case that the symptomatology, the clinical course, and the prognosis of cylindromas of the esophagus is not unlike that of the usual epidermoid cancer. The treatment also remains the same.

Summary

A complete review of the literature on the subject of primary adenocarcinoma of the esophagus is attempted. The second case of pure adenoidcystic carcinoma of the esophagus is presented. The symptoms, prognosis, and therapy of these tumors are similar to those of the usual squamous-cell carcinoma of the esophagus.

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Addendum

We have seen recently another instance of cylindroma of the esophagus at the middle third. The gross and microscopic features are similar to the case presented. The patient is alive and apparently well two weeks after esophageal resection. We consider this as the third authenticated case of adenoidcystic carcinoma of the esophagus.

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Five-Year Analysis of External Ethmoid and Frontal Sinus Operations

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The following cases of external frontal and ethmoid sinus operations, performed at the Manhattan Eye, Ear and Throat Hospital since Jan. 1, 1952, are presented in an effort to discover any information from a statistical study which may be useful in future diagnosis and treatment of patients with similar complaints. These cases were selected from operative records; therefore, each case reviewed has had one or more external sinus operations performed over this period. Not included in this group were those patients with malignant tumors that may have involved the frontal and ethmoid sinuses primarily or secondarily. Of the 50 cases presented, 32 are of infectious origin, either acute or chronic sinusitis. The remaining 18 cases consist of 17 frontal sinus mucoceles and 1 case of frontal osteoma. The analysis of these external operative cases, by year, is presented as follows:

	1952	1953	1954	1955	1956	1957	Total
Mucocele	3	2	4	1	1	6	17
Acute sinusitis	4	0	4	1	2	5	16
Chronic sinusitis	0	4	4	3	3	2	16
Osteoma	0	0	1	0	0	0	1
Total	7	6	13	5	6	13	50

The cases of mucocele and sinusitis and the osteoma of the frontal bone are taken from the otolaryngology clinic admissions of the Manhattan Eye, Ear and Throat Hospital and the private patients who were hospitalized with similar complaints leading to operation. Different surgeons, residents and attending staff, participated in the cases reported. The hospital admissions in the otolaryngology department for any one year vary between 6,000 and 7,000; the clinic outpatient visits number between 35,000 and 40,000. A follow-up questionnaire was sent out, and 32 responses were received.

The 17 cases of mucoceles included in this study may be considered a particular variation of chronic sinusitis. The etiology of the mucoceles that occur in the frontal sinuses is a combination of sinusitis infection and blockage of the nasofrontal duct, preventing adequate drainage. Contributing factors for this blockage that have been cited are trauma, allergy, and operative procedures. The exact etiology and predisposing causes, however, may never be discovered in a particular case.

The clinical course of the mucocele is chronic. It increases in size; consequently the bony walls of the sinus may be destroyed by pressure necrosis. Symptoms only may be produced when structures in the orbit, nasal cavity, or anterior cranial fossa are compressed. An acute reinfection of an existing mucocele can simulate acute frontal sinusitis, with severe pain and a febrile course. Intensive antibiotics, with supportive therapy and possibly operative drainage, are required at this time.

Mucoceles are secreting cysts lined with respiratory mucosa that will erode the bony sinus walls by pressure necrosis and eventually will extend to involve adjacent structures. The thick mucoid secretion which fills the cyst arises from the goblet cells and seromucinous glands that are present. Mucoceles of the frontal sinus also contain chronic hypertrophic inflammatory fibrous

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tissue and desquamation products of the chronic infection. Repeated infections in the presence of nasal allergy increase the growth and size of these mucoceles by causing more secretions and by obstructing drainage.

The 17 mucoceles in this series presented the characteristics described above. X-ray studies revealed in all cases a cloudy frontal sinus, with destruction of limiting sinus walls. Involvement of the frontal sinus was most frequently unilateral. The clinical picture was chronic in 11 cases and acute in 6 cases. The Lynch external frontal sinus procedure was performed in all of the 17 cases. There were 16 primary cases and 1 tertiary in this group; all cases had complete recovery. The patency of the nasofrontal duct for drainage was accomplished by a variety of methods: in six cases, no drain was used; in eight cases, a hard rubber catheter was used; in two cases, a Penrose drain, and in one case, a polyethylene tube. In the mucocele that was operated upon for the third time, no drain was used.

Contributing causes for intranasal obstruction with subsequent mucocele formation were found to be, in order of their incidence, as follows: (1) intranasal operative procedures; (2) trauma, gunshot wound, auto accident, and (3) unknown causes. To our knowledge, none of the primary mucocele cases required subsequent operative procedures for obstruction or recurrence.

No statistics are presented for patients admitted with sinusitis complaints that did not require surgical treatment. The operation performed most commonly (29 cases) was the Lynch external frontal sinus procedure, where an effort was made to preserve as much as possible of the anterior wall of the frontal sinuses, depending upon the pathology found. The frontal sinuses were entered inferiorly at the floor by way of the lamina papyracea of the ethmoid capsule. Additional procedures were commonly done, such as ethmoidectomy, sphenoidectomy, submucous resection, and internal and external maxillary sinusotomy, in order to facilitate the completeness of the sinusitis procedure in regard to the exenteration of disease tissue, drainage, and postoperative care. One bilateral partial ablation of the frontal sinuses was performed in a patient with a frontal lobe abscess. Two acute sinusitis cases in children required only external ethmoidectomy; one child was 2 months of age; the other, 9 years old, had small partially developed frontal sinuses. In no case was the classical Killian operation performed in order that the bone at the orbital margin was preserved for cosmetic purposes. No trephine operations were performed in this series.

The method of drainage intranasally by the nasofrontal duct, which was widely opened by these external operations, was as follows: No drain was used in 14 cases; a hard rubber catheter, in 15 cases, and a Penrose drain, in 3 cases. In no case was the acrylic or Lucite obturator used, nor was a split-thickness skin graft or mucous membrane flap employed, as has been described by others.

In 1 patient, an external frontal operation was performed for the third time, and in 10 patients the operation was secondary, varying in time of recurrence of symptoms from 2 to 22 years. Twenty-one sinusitis cases were initial external operations (osteoma excluded) that have not, to our knowledge, required a secondary external operation. One intranasal procedure was done subsequently for drainage in these 21 cases, while 2 cases had a recurrence of the sinusitis, which subsided with the use of antibiotics.

There were 16 acute sinusitis cases in this series, 2 females and 14 males. These acute sinusitis cases had the signs and symptoms of fever, headache, elevated WBC, periorbital edema, and swelling, together with tenderness and swelling over the frontal sinus. In many cases the frontal bone had periosteal irritation only, resulting in pain on pressure. In the severer acute

cases the edema was manifest, with bone involvement of the anterior wall of the frontal sinuses. Generally in all cases the eye was closed with induration and swelling of the lids. The time of onset of these acute cases was a period of 2 to 28 days prior to operation. Often there had been a history of cold or "flu" that contributed to the final outcome. Six of the sinusitis cases classified as acute were secondary operations, done because obstruction, together with reinfection of the frontal sinuses, produced the acute manifestations. The only death in this series occurred in the secondary group. This was a patient who developed a frontal lobe abscess who had had a bilateral partial ablation procedure in 1955, with recovery. Death followed a secondary attempt, in 1957, to evacuate the recurrence of the frontal sinusitis with this complicating frontal lobe abscess. The method of maintaining intranasal drainage was as follows: In 10 cases a hard rubber catheter was used as an intranasal drain; in 2 cases a Penrose drain and in 4 cases no drain was employed. X-ray evidence confirmed the clinical impressions in these cases. Cultures taken revealed most commonly Staphylococcus pyogenes var. albus and aureus, coagulase positive, with one case each of Diplococcus pneumoniae, viridans Streptococcus, and Pseudomonas aeruginosa.

Many of the S. pyogenes var. aureus cultures were of the hemolytic variety. Every case received some form of antibiotic or chemotherapy. Pathologic report revealed that chronic hypertrophic inflammatory tissue was present with edema in the lamina propria, with some polymorphonuclear leukocytes and many lymphocytes and plasmacytes; however, little fibrous tissue had developed. The pathology report did not always correspond to the clinical findings that classified the cases as acute sinusitis, and the pathology seen in microscopic sections is not as marked as the clinical symptoms would indicate in many cases.

There were 16 chronic sinusitis cases in this series: 2 females and 14 males. In this group the distinguishing element was duration and repetition of symptoms over a period of years, marked by frequent associated intranasal procedures, such as ethmoidectomy, sphenoidectomy, submucous resection, and Caldwell-Luc procedures, in an effort to irradicate the sinus infection and obtain drainage before resorting to an external operation. Headaches, recurrent and infrequent, together with tenderness over the frontal sinuses, were the usual symptoms. X-rays showed an involvement of associated sinuses usually unilaterally. As is often the case, the WBC was normal and the temperature in the majority of cases was not elevated. The allergic factor in these cases was not determined, although clinical evidence of polypi and increase in symptoms in the allergic season pointed to its importance. Four cases in this group of sixteen were secondary operations, the remainder primary. A hard rubber catheter was used for intranasal drainage in only 5 cases, no drain in 10 cases and a Penrose drain in 1 case. Unfortunately, cultures were generally not taken for organisms and sensitivity at the time of operation. Every case received some form of antibiotics or chemotherapy. The pathology reports revealed chronic hypertrophic inflammatory tissue, with infiltration of lymphocytes and plasmacytes and often eosinophils. Fibrous tissue was commonly present, with moderate inflammatory reaction noted in these chronic sinusitis cases.

There is one case in which osteoma of the frontal sinus was removed during this series. At operation removal was accomplished without incident, and microscopic sections, by Dr. Andrew A. Eggston, confirmed the clinical and x-ray diagnosis.

The follow-up results in the 32 cases of acute and chronic sinusitis have revealed that 1 patient was operated on, for the third time, in January, 1958, having an external frontal operation on the opposite frontal sinus from the initial two operations. This is the only case of the series that has required an additional external operation. Frequent complaints of headache after op-

eration were revealed in the questionnaire, but they did not persist and gradually subsided. One intranasal procedure was done for drainage in a primary case, as stated before, and a secondary case also required an additional drainage procedure in 1958. Two primary cases required antibiotics owing to a recurrence of the sinusitis, but operation was not required. One patient complained of double vision. One death occurred in the acute sinusitis group after the secondary operation from a frontal lobe abscess and meningitis. No response or information was obtained from 18 patients.

Complications found in this series included the frontal lobe abscess resulting in meningitis and death. One drug reaction to chloramphenicol (Chloromycetin) and one penicillin reaction occurred. Three of the patients operated upon had an external draining fistula from the frontal sinus prior to operation: one, a 2-month-old infant; the second, a 37-year-old man, and the third, a 12-year-old boy. In one female, a wound infection occurred, probably because of lack of drainage through the nasofrontal duct. The hospital stay for the clinic patients was generally longer, averaging 18 days against 15 days for the private patients.

Comment

There were 28 males and 4 females in the sinusitis series, clearly indicating the sex predilection. The age varied in these cases from an infant of 2 months to an adult of 66 years, with a mean age of 39. After the surgery a hard rubber catheter was used as a means of maintaining the nasofrontal opening oftener in the acute cases than in the chronic cases. The table below lists the methods employed for maintaining the nasofrontal opening.

	Rubber Catheter	Penrose	None	Total
Acute sinusitis				
Primary	6	0	4	10
Secondary	4	2	0	6
Total	10	2	4	16
Chronic sinusitis				
Primary	3	1	8	12
Secondary	2	0	2	4
Total	5	1	10	16

All cases presented received some antibiotic or chemotherapeutic agent or both. The benefit of the use of these therapeutic agents separately cannot be evaluated, since all cases received a combination of antibiotic and chemotherapy. In the yearly incidence of acute and chronic sinusitis requiring an external operation there seems to be no correlation between the more widespread use of chemotherapy and any decrease in the incidence of cases. The infrequent complications attributed to external frontal operative procedures and the therapeutic benefits obtained indicate their merit and value in the management of frontal sinusitis.

Conclusions

In this series males predominated over females in occurrence of sinusitis requiring operation.

The hard rubber catheter was found to be more useful in maintaining the patency of the nasofrontal duct, especially in the acute sinusitis cases.

The major contributing cause of nasofrontal duct obstruction with subsequent mucocele formation in this series was found to be intranasal operative procedures.

The yearly incidence of acute and chronic sinusitis that requires external operation has not been altered by the more intensive use of the antibiotics.

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Benign Hypertrophy of Masseter Muscle

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Since Legg,[13] in 1880, reported a "tumor of ill defined outline" involving the masseter and temporalis muscles in a 10-year-old girl, less than 40 cases of benign masseter hypertrophy have been described in the literature. However, the fact that the 3 cases discussed here came under our observation during a period of less than 7 months, together with Barton's[2] statement of having seen more than 30 cases within 10 years, indicates clearly a more frequent incidence of this condition. According to Caldwell,[4] the paucity of reports may be due to its comparatively small clinical importance, except in differential diagnosis and in instances where there is an indication for surgical correction for cosmetic reasons.

Though the etiology of benign masseter hypertrophy has not been determined with finality, several theories of its origin have been advanced. Barton[2] describes two types which may be unilateral or bilateral. In the congenital or familial type the patients have a "chipmunk look," because the muscle stands out in a thin or bony face. The auricles appear closely adherent to the head. Patients with the commoner, or acquired, type are tense and habitually clench or grind their teeth in sleep. Malocclusion or secondary wearing down of teeth surfaces may be found. In unilateral cases, which are often of striking appearance due to the asymmetry, some teeth may be missing, requiring chewing on the hypertrophied side only. Similar explanations are given by Boldt[3] and Thoma.[21] But Adams,[1] Masters et al.,[14] and Maxwell and Waggoner[15] do not agree with this theory of work hypertrophy. Tempest[20] states that in unilateral cases temporomandibular joint derangements, bad dental occlusion, or addiction to chewing gum may be postulated as possible causes. But he emphasizes the fact that in actual practice this association is hardly ever seen.

In specimens obtained by biopsy or at operation normal striated muscle fibers, with an occasional increase of nuclei, are seen. Boldt[3] found enlargement of some muscle fibers. Maxwell and Waggoner[15] reported an increase in size of some of the muscle fibers, with lack of striation. They considered these findings of questionable significance. Wolhynski[22] differentiates three periods of the normal masseter muscle: growth from the first anlage to age 20, relative stability from age 20-35, followed by gradual degeneration. It is therefore not astonishing that the age of nearly all persons with masseter hypertrophy falls within the second to fourth decade of life. The cases are rather equally divided between men and women.[11] Approximately half the reported cases were unilateral and half bilateral. With the exception of two Negroes[10,19] all patients belonged to the white race, including one East Indian.[7]

Trauma plays no role in the development of the hypertrophy. The onset of the acquired form usually is gradual, over a period of several months. Very often the patient himself is unaware of the painless swelling, but his attention is called to it by some friends. The growth might subside spontaneously but usually recurs rapidly.[19]

The majority of patients have no actual complaints except fear of a developing malignancy. Some noticed the swelling only because they were asked about it[10]; others

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thought they had mumps. A few people with malocclusion complain of discomfort or easy fatigability on prolonged chewing. But trismus is absent.

Nearly all patients are diagnosed as having a disease of the parotid gland. The following diagnoses have been made before the true nature of the growth was discovered: parotitis (specific or nonspecific), mixed and other parotid tumors, salivary stones, and retention cysts[6] in the parotid gland; other tumors, such as lipoma, rhabdomyoma, lymphangioma, and sebaceous cysts, have been thought of, but even diagnoses of otitis externa, temporomandibular arthralgia, and dental diseases have been entertained as a cause of the swelling. Very often the actual masseter hypertrophy is discovered only during operation for one of the assumed diseases.

In diseases of the parotid gland the swelling is usually nearer to the ear lobe.[10] On palpation the bundles of the contracted masseter muscle bulge under a freely movable normal skin. There is free flow of saliva through a normal opening of Stensen's duct.

Neurological examination and electromyography are generally inconclusive.[15,17] Benign masseter hypertrophy must also be differentiated from actual involvement of this muscle. Cases of syphilis,[3,6] tuberculosis, trichinosis, and actinomycosis and two cases of hydatid cysts are quoted by Coffey.[6] The same author mentions that only nine cases of primary tumors of the masseter were reported in the literature, namely, fibrochondroma, rhabdomyosarcoma, cystic sarcoma, and angioma. These conditions can usually be ascertained by the appropriate means. The few cases of traumatic myositis ossificans[3,5,16] in the masseter muscle are readily recognized by their x-ray appearance.

Lateral "flaring" of the inferior border of the mandible may be caused by the abnormal tension from the muscle. The comprehensive study of Maxwell and Waggoner,[15] however, calls attention to the fact that a person may have a very wide flaring mandibular angle without masseter enlargement or may have the muscle enlargement associated with mandibles of normal shape.

We found regularly in our patients with benign masseter hypertrophy a laterally displaced Stensen duct in the presence of normal filling of the parotid gland "tree." While this displacement is readily visible in the frontal view, it may not always appear in the lateral sialogram. These sialographic findings were associated with a constantly present exostosis of the mandibular angle on the affected side.

This "spine-like projection" of the angle of the mandible, forming an integral part of the insertion of the hypertrophied masseter muscle, was also observed in all cases of Adams[1] and Masters et al.[14] (during operation) and by Maxwell and Waggoner[15] (x-ray evidence). It may be explained by irritation of the periosteum at the mandibular angle by the action of the masseter muscle, resulting in additional surface for the insertion of muscle fibers.

Therapy consists essentially in reassurance, correction of faulty habits of mastication, and, of course, restoration of a normal bite by prosthesis, if necessary.[2,4,8,17] Surgical excision of parts of the muscle, with or without inclusion of some of the protruding angles of the mandible, has been performed,[1,9,14,18,19] sometimes with complete personality change in a disturbed person.[19]

Figure 1 (Case 1)

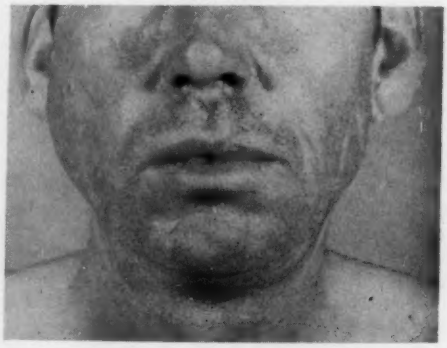

Fig. 2 (Case 2).—Note exostosis at angle of mandible.

Report of Cases

CASE 1.—A 50-year-old white man was admitted Dec. 17, 1957, with diagnosis of adenoma of the thyroid gland, for which he was operated on. Patient was noted on routine clinical examination to have hypertrophy of right masseter muscle. This was verified by parotid sialography. He had no symptoms referable to this condition. Teeth were in poor repair, with two upper left incisors missing. During hospitalization patient had extraction of No. 26 for periapical pathosis, and an upper partial denture was constructed. No further treatment was recommended (Fig. 1).

CASE 2.—A 34-year-old white man was admitted on the neurosurgical service, on April 23, 1958, with diagnosis of atypical facial neuralgia. He was treated with histamine, without significant improvement. He complained of pain originating just in front of the left ear, radiating to the left mandible and orbital ridge and temple. He had constant dull pains for several months preceding admission. The patient's left jaw had been swollen and larger than the right jaw since 1944. He avoided chewing on the left side for fear of precipitating attack of pain. Ear, nose, and throat examination was within normal limits. Dental examination was negative; the mandible had no limitation of motion. Parotid sialography revealed lateral displacement of the parotid duct and exostosis at the angle of the mandible (Fig. 2).

CASE 3.—A 32-year-old white woman was admitted elsewhere, on July 15, 1958, with diagnosis of possible left parotid tumor, probably mixed. The patient had a painless lump of the left jaw of approximately three months' duration. Some dull pain about the left ear and teeth was elicited for a few weeks prior to admission. Examination revealed a circumscribed, nontender mass overlying the angle of the left mandible. The remainder of the physical examination was within normal limits. Left parotid sialography revealed a normal gland, lateral displacement of the parotid duct, and exostosis of the angle of the mandible (Fig. 3). Operation, performed elsewhere, revealed the tumor mass to be in front of the parotid gland and to consist of the masseter muscle. The parotid gland was grossly normal. A biopsy specimen was taken from the parotid gland and masseter muscle. Histological examination revealed no significant abnormalities.

Fig. 3 (Case 3).—Note exostosis at angle of mandible.

Summary and Conclusions

Various aspects of benign masseter hypertrophy are discussed, with reports of three new cases. The chief problem is one of correctly diagnosing this swelling. A triad consisting of (1) a growth in the masseter region; (2) sialographic demonstrations of a laterally displaced Stensen duct in the presence of normal filling of the parotid gland, together with (3) x-ray evidence of an exostosis at the mandibular angle, will help to establish the diagnosis.

Dr. Bourbon E. Canfield, Instructor in Surgery, and Dr. Lawrence A. Davis, Associate Professor of Radiology, University of Louisville School of

Medicine, gave us permission to publish report and x-rays, respectively, of Case 3.

Veterans Administration Hospital (2).

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The Responsibility of the Speech Therapist to the Laryngectomized Patient

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I. Introduction

The incidence of total laryngectomy is definitely on the increase both in the United States and abroad. Martin[1] reports that at least 1,315 laryngectomies were performed in the United States in 1952 compared to 846 in 1947. This represents an increase of 55%.

These patients require the services of personnel trained in methods of speech rehabilitation for the laryngectomized. Equen[2] has stated that "the laryngectomy without rehabilitation may serve only to prolong a miserable existence." The methods of speech production available to the laryngectomized patient include esophageal speech or the use of some type of artificial larynx. Esophageal speech is the method of choice for a majority of patients who have undergone the procedure and instruction in this method of voice production is offered in many institutions. There are some patients, however, who are unable to master esophageal speech even after an intensive program of therapy. These patients must be given a means of communication and, for some of them, one of the artificial larynges may represent a practical solution. The speech therapist working with these patients must be familiar with the different types of artificial larynges and be able to utilize them in a program of rehabilitation designed to give each laryngectomized patient a means by which he can communicate with others.

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II. Description and Limitations of Artificial Larynges

A. Mechanical Larynx.—This is a metal cylinder containing a reed which serves as the vibrator. Two flexible rubber tubes extend from the cylinder, one from the top and one from the bottom. The lower tube is placed into the tracheal stoma. The upper tube goes into the patient's mouth. There is a small opening in the cylinder which permits breathing through the apparatus when the patient is not speaking. By covering the opening in the cylinder, the air coming up through the bottom tube from the tracheal stoma sets the reed into vibration and a sound similar in quality to that of a toy horn is produced. This sound then goes through the top tube and into the mouth where it is formed into words by the normal articulatory movements of the lips, teeth, tongue, etc.

The instrument is relatively inexpensive (current price is $15.50) and is manufactured by the Western Electric Corporation. Distribution of this type of artificial larynx is handled by the Bell Telephone Laboratories. Physicians and therapists who wish to make arrangements for the purchase of this type of artificial larynx for their patients may contact the medical department of their local telephone office.

The *disadvantages* of the artificial larynx are as follows:

1. The method is unsanitary. The insertion of a rubber tube into the tracheal stoma may interfere with the proper hygiene of this area. The tube inserted into the patient's mouth may serve as a possible source of infection if it is not properly and frequently cleaned.

2. Compensatory adjustments of articulators. Because one tube is always in the mouth when this type of artificial larynx is used, normal articulation is difficult. Compensatory adjustments of the articulators are required in order that the patient may produce intelligible speech while the tube is in the mouth.

3. Limitation of use of one hand. The instrument is conspicuous and must be held in the hand. Therefore, physical activity is somewhat limited. This is a particular problem for patients whose work involves the use of both hands, e. g., a salesman demonstrating equipment to a customer.

4. Saliva tends to accumulate in the tube which is held in the patient's mouth. When this occurs, the instrument will not operate.

B. Electrolarynx.—This is an electric vibrator which is held by the patient against the side of the neck and is similar in sound to an electric razor. The walls of the neck are set into forced vibration. The buzzing sound produced is complex, nonperiodic, and inharmonic. The cylinder of the conventional electrolarynx is 4 in. in length and 1 in. in diameter and it is battery powered. The initial cost is about $150.

The Aurex Corporation has recently developed and will soon have available for distribution an improved "all-in-one" version of their Electrolarynx. The new instrument has both the vibrator and the battery in one compact unit which includes a volume control. The initial cost is slightly higher than that of the presently available model and the operating cost is appreciably higher.

The disadvantages of the electrolarynx are as follows:

1. The instrument is conspicuous and requires one hand to operate.

2. The initial cost of the instrument is relatively high and batteries must be periodically replaced. The battery used in the present instrument costs about $1.80 and lasts an average of five to six weeks.

3. The voice quality is monotonous and no change of pitch is possible.

4. There is considerable ambient noise produced when the instrument is used which can be controlled, however, to some extent by skillful use of the instrument.

One of the newer modifications of the electrolarynx is the Cooper-Rand Electronic Aid (Rand Development Corporation, Cleveland). This instrument permits some changes of pitch and volume by the patient. The insertion of a tube into the patient's mouth is required when the instrument is used. This tube conducts the sound from the amplifier into the user's mouth. Compensatory adjustments of the articulators are therefore required as with the mechanical larynx. Saliva tends to accumulate in this tube and, when excessive, will interfere with the function of the instrument.

III. Indications for the Use of the Electrolarynx

It is generally agreed that the most satisfactory method of voice production for the postlaryngectomized patient is esophageal speech. This method of voice production is being taught to laryngectomized persons in institutions throughout the country. In the present paper, some of the applications of the electrolarynx to the speech rehabilitation of patients who have had a total laryngectomy will be described.

Levin[3] enumerates several indications for the use of the electrolarynx:

(1) Stenosis of the esophagus after extensive removal of a widespread malignant lesion; (2) resection of the cervical portion of the esophagus, (3) multiple handicaps, for example, laryngectomy plus deafness, (4) suspected recurrence, metastasis, or multiple lesions, for example, a bronchial carcinoma in addition to the laryngeal lesion, (5) senility or other feebleness.

I wish to suggest several additional applications for the use of the electrolarynx.

1. *To provide increased volume for the patient in specific situations in which esophageal speech is unsatisfactory.* There are many patients who have developed excellent esophageal speech which is satisfactory for a majority of person-to-person conversational situations. However, in a noisy restaurant, a crowded street, or when talking to a group where amplification is

not available, these patients report that their usual mode of speech production does not provide sufficient volume. It is suggested that in these specific situations the patient be permitted to use the electrolarynx. One patient, for example, finds considerable difficulty addressing a group of workers in a noisy shop, but has little difficulty in other situations. The electrolarynx is for him an excellent supplement to the method of speech production which he uses in a majority of speaking situations.

2. *To permit the patient to communicate with the therapist and with other patients in the early stages of esophageal speech instruction.* The initial therapy sessions are frequently a frustrating experience to the laryngectomized patient who wishes to communicate with his therapist, but who is unable to do so because he has not yet developed esophageal speech. Usually the patient resorts to the use of pencil and paper at this time. This tends to limit communication with the therapist at a time when it is most important. It is awkward and time-consuming for the patient to write several sentences or paragraphs to the therapist. It is also cumbersome to transmit this material to other patients if therapy is being administered in a group situation. The use of the electrolarynx permits communication at this stage between the patient and other persons in the therapy situation. It is my opinion that the use of the electrolarynx for this purpose will not serve to diminish motivation for the development of good esophageal speech since the electrolarynx at best is a poor substitute for esophageal speech production.

3. *To assist laryngectomized persons having difficulty on the telephone.* Some laryngectomized persons find that esophageal speech is not satisfactory on the telephone. The results with the electrolarynx are very satisfactory because ambient noise from the instrument is not transmitted over the telephone. For laryngectomized persons, therefore, who are having particular difficulty on the telephone, the electrolarynx is of benefit to them while they continue using esophageal speech for most speaking situations.

4. *Use by patients with hypopharyngeal or tracheoesophageal fistulas.* Patients with fistulas are sometimes advised by their surgeons not to attempt esophageal speech because the pressure built up in the hypopharynx may increase the flow of secretions from the fistula or may interfere with the healing of a recently repaired fistula. The electrolarynx will not be contraindicated in these cases because no trapping of air and building up of pressure is involved.

5. *Use during periods of physical or emotional distress.* The electrolarynx can temporarily be used when the patient has a cold or is experiencing a period of emotional distress which may interfere with the production of esophageal speech.

6. *Use by laryngectomized patients with temporary or permanent colostomy.* One patient treated at a local institution had had a colostomy prior to the laryngectomy. The intra-abdominal pressure involved in the production of esophageal speech caused incontinence. Attempts at the development of esophageal speech had to be abandoned, and the patient was taught to use the electrolarynx.

7. *Use by patients unable to master esophageal speech.* The electrolarynx should be introduced by the therapist when the patient is unable to master esophageal speech satisfactorily. The responsibility of the speech therapist to the patient does not end if the patient is unsuccessful in developing esophageal speech. It is his responsibility to provide a means of communication for laryngectomized patients who are unable to master intelligible esophageal speech.

(*Use by nonlaryngectomized persons during periods of complete voice rest.* The electrolarynx may be used by nonlaryngectomized persons during periods of complete voice rest. Laryngologists frequently advise voice rest for the patient with acute laryngitis to prevent the development of a localized area of irritation and consequent pathology which may result from continued

voice use. Voice rest is also indicated following surgery for the removal of benign lesions of the larynx, such as vocal nodules or polyps, in order to facilitate complete healing of the operative area. During periods of voice rest, whispering is also contraindicated because this type of speech production involves the approximation of the vocal folds. This is particularly true of the tensed vocal cord approximation characteristic of the "stage whisper" which many patients begin to use after being placed on voice rest. The use of the electrolarynx is not contraindicated when the patient is on complete voice rest because no approximation or movement of the vocal cords is involved. The only movements required are the normal articulatory adjustments of structures above the larynx.)

IV. Failures in Esophageal Speech Therapy

It is difficult to determine the percentage of laryngectomized patients who do not develop satisfactory esophageal speech. McCall [4] reviewed 149 laryngectomized patients, reporting on 126, of whom 67 "developed a good voice and 13 were able to learn the act." Forty-six did not learn and twenty-three were not reported on. Pitkin [5] reported 61 cases, of which at least 21 did not learn to speak. Thirty-five returned to their work and three did not answer the questionnaire. Heaver, White, and Goldstein [6] report on 274 laryngectomized patients admitted to the National Hospital for Speech Disorders. The authors state that 110 patients, or 40.1%, discontinued treatment against advice. The authors state:

> Although one-third of this group had to cease their attendance because of such factors as the need for additional surgery, medical or serious emotional complications, generalized weakness, travel distance, or demise, the remaining two-thirds apparently left the clinic because of overwhelming feelings of futility regarding the possibility of ever speaking as adequately as they once did. Their subjective estimate of what constituted improvement was differently based than the objective estimate of the therapist.

Possible conclusions from these and other studies are as follows:

1. Approximately one-third of all patients do not learn esophageal speech.[7]

2. It is possible that patients dropping out of therapy programs are not adequately followed to determine the reasons for their failure. Some of these patients may achieve eventual success at an institution other than the institution which saw the patient initially. Some of these patients may resume therapy months after an initial unsuccessful attempt at developing esophageal speech. Follow-up studies of laryngectomized patients who drop out of therapy programs are inadequate. Studies reported, therefore, may not do justice to the number of patients who eventually master esophageal speech satisfactorily.

3. It is possible that therapists having the greatest percentage of success with patients are not reporting their results. (Are their therapy schedules so heavy that they do not have time to analyze and report their results?)

Conley, DeAmesti, and Pierce [9] recently described a surgical procedure to assist the laryngectomized patient in the mastery of esophageal speech. It consists of creating a mucosal tunnel through the wall of the cervical esophagus, causing a controlled communication between the trachea and gullet. The tube is constructed in such a manner that air can pass into the esophagys without the disadvantage of food and saliva passing onto the neck or into the trachea. The authors report that following this surgical procedure, patients can, with simple instructions, usually speak on the first effort. This procedure may enable a larger portion of laryngectomized patients to develop esophageal speech and should be carefully evaluated by laryngologists and speech therapists.

V. Who Should Decide When the Use of the Electro Larynx Is Indicated?

It is the current practice of the postlaryngectomy speech department of the National Hospital for Speech Disorders to demonstrate the electrolarynx routinely to

the new patient at the time of his first therapeutic session. The patient is told that he may use the electrolarynx immediately if he chooses, or, if he prefers, he may have esophageal voice therapy for four to six weeks without the instrument to establish a fair trial to determine his capacity to learn and use the new voice, or, finally, if his economic status and job are in immediate jeopardy, he may concurrently take voice lessons and use the instrument while at work. Whatever choice is made is entirely up to the *patient*.[8] Lueders[7] believes that the laryngologist who has performed the surgery should decide which patients cannot learn to speak by the esophageal method.

I believe that the attention of the *speech therapist* should be directed toward the development of a means of communication by the laryngectomized patient. We dare not be committed to one method of speech production alone. *The speech therapist, working in conjunction with the surgeon,* should decide which patients are unable to develop satisfactory esophageal speech. This decision should be reached only after the patient has been exposed to an intensive period of esophageal speech instruction. A minimum of three months of training should be given to the patient before it is decided that a substitute method of speech production is necessary. Only after such a rigorous program of therapy should the decision be reached that a particular patient must learn an alternate method of voice production.

When the patient drops out of the therapy program against the advice of his therapist, the referring surgeon should be immediately informed. The cooperation of the patient's surgeon should be enlisted at this time in an effort to determine the reasons for the discontinuance of therapy. Every effort should be made in this situation by the surgeon and the therapist to investigate and, when possible, resolve these problems so that eventual success in speech rehabilitation may be achieved.

VI. Suggestions for the Program of Speech Rehabilitation

Many therapists who are responsible for the speech rehabilitation of postlaryngectomized persons are classified by their institutions as "esophageal speech teachers." Since esophageal speech is only one means of communication available to the laryngectomized patient, it is suggested that the term "postlaryngectomy speech therapist" be used. The job of the therapist is to give the patient a method of communication. Esophageal speech is one method used to accomplish this end. To refer to a postlaryngectomy therapist as an "esophageal speech teacher" is somewhat analogous to a therapist working with stutterers classifying himself as a "Johnsonian speech therapist" or one who works with lispers as a "Froeschel's 'f' method therapist." The method is only a means to the end, and the end is the development of the most efficient method of communication possible for a given patient. To incorporate a particular method in the title of the therapist commits the therapist to some extent to the development of that particular procedure. The term used at one institution for teachers of postlaryngectomized patients is "alaryngeal teacher." This term at least does not commit the therapist to a particular approach to his postlaryngectomized patient. However, it seems undesirable to describe the activity of the therapist in the negative sense, i. e., alaryngeal refers to without the larynx. The term, "alaryngeal" in this sense means the therapist is without a larynx.

Research is needed to investigate the factors that account for the noncooperation by some laryngectomized patients in speech rehabilitation programs and the reasons that they drop out of therapy. A clinical psychologist on the rehabilitation team might reduce the number of patients who are unsuccessful in developing esophageal speech. A psychologist would certainly be helpful in investigating the reasons for the

failure of some patients to complete a course of speech therapy.

VII. The Goal of the Postlaryngectomy Speech Therapist

The speech therapist should use all the means at his command to achieve the best possible esophageal speech for the patient. However, his responsibility to the patient is to provide for him a workable means of communication. Therefore, he cannot be committed to one form of voice production alone. The electrolarynx should be part of the armamentarium of the postlaryngectomy speech therapist. He must know when to introduce it and how it should be used. He must not feel his job is over when the patient, after an intensive period of esophageal speech instruction, fails to master the method satisfactorily. An efficient means of communication must be the goal of the therapist for each laryngectomized patient.

I wish to express my appreciation to Dr. I. A. Polisar for his assistance and suggestions in the preparation of this manuscript.

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Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

The Effect of Phenylephrine (Neo-Synephrine) on Pure-Frequency Audiometric Scores

E. THAYER CURRY, Ph.D.; FLOYD BOYS, M.D., and DONALD B. STONE, M.S., Urbana, Ill.

General

This study is concerned with the effect of temporary closure of the Eustachian tube on hearing acuity scores obtained during pure-frequency audiometric measurement. The "common cold," nasal allergies, and tobacco smoking with inhaling presumably could produce some edema of the orifice of the Eustachian tube, resulting in partial closure, and thus have direct effects on the auditory threshold as measured by the pure-frequency audiometer. It is not unreasonable to expect hearing scores to be slightly less sensitive even when there is a slight closure of the Eustachian tube from some of the common forms of congestion mentioned above.

Problem

These considerations motivated us to conduct a simple preliminary study on the effects of the use of a nasal spray on hearing acuity scores, the spray presumably keeping patent or open the orifice of the Eustachian tube. In undertaking this study, we proposed to assess the hearing of the subjects both before and after the use of the nose spray.

Submitted for publication Dec. 15, 1958.

E. Thayer Curry, Professor of Speech and Director of the University Hearing Center, University of Illinois, personally conducted the audiometric measures of this study. Dr. Floyd Boys, Associate Professor of Health Education, supervised the Master of Science Dissertation, on which this report is based. Donald B. Stone prepared the substance of the Master of Science Dissertation.

Procedure

The solutions used were as follows: (1) a 0.5% phenylephrine (Neo-Synephrine) solution to be used as a nasopharyngeal spray (experimental solution); (2) an isotonic saline solution to be used as nasal irrigation (control solution). The auditory thresholds of normal-hearing subjects were assessed with pure-frequency audiometric stimuli at 500, 2,000, and 8,000 cps.

Male college students (aged 18-26) whose hearing acuity showed no greater than a 5 db. hearing loss were selected as subjects. Certain individual controls were required of the subjects to insure that the test was standardized as to health, diet habits, tobacco usage, etc. No subject was tested who had a "cold" or upper respiratory infection within one week prior to the time of his appointed hearing measurements. Each subject had agreed not to smoke on any given test day until the conclusion of the hearing measures.

Five subjects, two nasal solutions, and three audiometric frequencies were used in this study; both ears of each subject were measured individually. The two test solutions were employed on separate test days. Some subjects were given the isotonic saline solution on the first test day and the 0.5% phenylephrine solution on the second test day. In other subjects, the order of administration of the solutions was reversed. The isotonic saline solution was administered with a Bermingham Glass Nasal Douche, each side of the nose being irrigated with the contents of one-half of the glass douche. The phenylephrine solution was administered by four or five bulb compressions with a standard De Vilbiss Atomizer at both the nasal orifices and the posterior nasopharynx through the mouth. Thus, each experimental subject provided 24 threshold measures; 120 threshold determinations make up the total data for this study.

The subjects were measured on a standard commercial audiometer, which had been recently acoustically calibrated at the factory with an artificial ear. The acoustical calibration was checked

TABLE 1.—*Hearing-Threshold Differences Before and After Use of Isotonic Saline Solution in Nasopharyngeal Passages* *

	CPS					
	2,000		500		8,000	
Subject	L	R	L	R	L	R
1	3	0	0	3	5	6
2	0	0	0	0	5	−3
3	5	3	−1	0	0	0
4	0	0	5	2	0	0
5	0	0	2	−5	0	0

* L indicates left ear; R, right ear. These threshold differences were measured in units of 1 db. (sound pressure).

against loudness judgments of a group of 10 persons with normal hearing. The instrument employed in this study, the Allison Audiometer (Model 20-A), measured sound-pressure values in 1 db. units. This machine met the specifications for audiometers stated by the Council on Physical Medicine and Rehabilitation of the American Medical Association and by the American Standards Association. All hearing measurements were conducted in the specially constructed, sound-treated rooms of the University Hearing Center of the University of Illinois.

Results

Table 1 presents a comparison of hearing threshold differences before and after use of the isotonic saline solution in the nasopharyngeal passages. Table 2 presents the same comparison for the phenylephrine solution.

It was the original intention to employ an analysis of variance and a *t*-test in the statistical evaluation of the data obtained in this study. However, later statistical consultation suggested that these two methods be abandoned. Instead, it was decided to employ just a *sign test*. A sign test is a simple statistical device that employs a system of listing experimental or other data in tabular form, noting the number of times that one thing is better than another. This simple statistical method of analysis was selected because the results obtained in this investigation seemed to be satisfactorily evaluated in this tabular form.

TABLE 2.—*Hearing-Threshold Differences Before and After Use of Phenylephrine Solution in Nasopharyngeal Passages* *

	CPS					
	2,000		500		8,000	
Subject	L	R	L	R	L	R
1	0	0	2	0	5	−10
2	0	0	5	−5	10	2
3	3	−1	0	0	0	0
4	0	0	−1	0	0	0
5	0	0	−4	−4	0	0

* L indicates left ear; R, right ear. These threshold differences were measured in units of 1 db. (sound pressure).

TABLE 3.—*Sign Test, Simple Comparative Analysis of Effects of Phenylephrine Solution Versus Isotonic Saline Solution on Thresholds of Hearing Acuity* *

Frequency Level, CPS	PE Better	No Difference	SS Better
2,000	0	7	3
500	4	1	5
8,000	2	7	1

* PE indicates phenylephrine, 0.5%; SS, saline solution (isotonic).

Table 3 presents a sign test comparative analysis of the effects of phenylephrine 0.5% solution versus isotonic saline solution on the thresholds of hearing acuity in the series of persons studied. Table 3 indicates that thresholds of hearing acuity were more sensitive following the use of phenylephrine 0.5% solution in only 6 of the 30 comparisons. On the other hand, following the use of isotonic saline solution the thresholds of hearing acuity were more sensitive in 9 of the 30 comparisons. In 15 of the 30 comparisons no hearing-threshold differences were observed regardless of the solution employed.

Comment

The threshold comparisons of this investigation would seem to warrant emphasis of the following points:

1. It is obvious that phenylephrine 0.5% solution had no outstanding effect on improving hearing acuity thresholds. This was somewhat surprising, as the investigators had predicted that the decongestant solution might have some effect on the patency of

the orifice of the Eustachian tube, thereby possibly causing an improvement in hearing-acuity scores.

2. On the other hand, irrigation of the nose and nasopharynx with isotonic saline solution was borderline in its action or effect on hearing acuity thresholds. The data possibly indicate some slight improvement in hearing-acuity scores when this inert, control solution was employed.

3. Thus, in view of the borderline results obtained from use of either 0.5% phenylephrine or isotonic saline solution, it may be that their value was mainly mechanical. That is, the solutions may have washed out mucus and exudate lying on the nasal mucosa and on or around the orifice or opening of the Eustachian tube, thereby keeping the orifice open. If this be true, with a more patent Eustachian tube the air pressure inside the middle ear possibly would be better equalized with that of the outside air in the nasopharynx and thus hearing would be more nearly normal.

Recommendations

1. It should be pointed out that the standard commercial audiometer employed was not sufficiently sensitive to check the whole range of hearing of the normal-hearing subjects used in this study. Commercial audiometers are designed primarily to assess the hearing of persons with hearing difficulties. The most sensitive limit on the sound-pressure scale of the study audiometer was −10 db. This limit was not sufficiently sensitive to measure any changes in hearing acuity below the limit value. Thus, in 15 of the 30 comparisons made, any change which may have occurred in these subjects was not measurable because of instrument limitations. In any repetition of this study an instrument with greater sensitivity would be mandatory.

2. It would seem worthwhile that this same type of study be repeated in our own or other laboratories but on a much larger scale. When the comparisons noted in the paragraph above are excluded, the present data are from too small a series of measures to evaluate accurately the value or effect of phenylephrine solution on hearing acuity scores. But the idea behind the investigation is considered sound and worthy of further study.

3. Since both of these solutions presumably might have some mechanical "flushing" value, as previously considered, it is very possible that a more thorough and rigorous employment of both nasal douching and spraying with just plain isotonic saline solution might have produced something of the effect predicted in this experimental study. Certainly, any repetitive study in this area should include this second recommendation.

4. This preliminary study employed only healthy subjects with normal hearing acuity. It is strongly advised that any new clinical study of this sort should include subjects with upper respiratory infections and nasal allergies (e. g., hay fever).

5. It would be possible to repeat the study with the use of nasal smears to ascertain the presence or absence of specific nasal mucosa pathology. Observation of bacteria and pus cells would be indicative of bacterial or viral infection of the nasal mucosa, while eosinophils would be suggestive of local mucosal allergy. Such additional examination should provide a more positive means of designating the normalcy of the person's nasopharyngeal mucosa at the time of the hearing examination.

6. After the administration of the phenylephrine solution, it might be desirable to check visually the patency of the Eustachian tube.

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Progress in Otolaryngology

Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Internal Ear

Summaries of the Bibliographic Material Available for 1955

BRUCE PROCTOR, M.D., Detroit
With the Collaboration of Michel Portmann, M.D., Bordeaux, France; Enrico Bozzi, M.D., Milano, Italy; Jerzy Szpunar, M.D., Krakow, Poland, and Thea A. Hennecke, M.D., Windsor, Ont., Canada

A review of the literature on a subject such as chronic progressive deafness is more valuable for reference if its scope is widened to include papers published in foreign languages. To this end we have obtained the collaboration of Dr. Michel Portmann (French), Dr. Enrico Bozzi (Italian), Dr. Jerzy Szpunar (Polish and Russian), and Dr. Thea Hennecke (German). A portion of the other language papers are abstracted from surveys published in "Excerpta Medica," the *Annuals of Otology, Rhinology and Laryngology,* and in the A. M. A. ARCHIVES OF OTOLARYNGOLOGY.

The various papers are abstracted to include new material pertaining to the subject of deafness. Occasionally more space is devoted to a scholarly presentation of old and familiar material, and so ultimately we include both new and old information presented in easily read and condensed form.

The material has been arranged in the following sequence: otosclerosis, Ménière's disease, reconstructive surgery for deafness, acoustic and head trauma, toxic labyrinthitis, inflamatory diseases of the labyrinth, deafness in children, intracranial pathology and deafness, medical treatment of deafness, miscellaneous contributions on deafness and tinnitus, vertigo, vestibular tests, anatomy of the ear, cochlear physiology, and vestibular physiology.

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Associate Professor of Otolaryngology, Wayne State University (Dr. Proctor); Professor Agrégé à la Faculté de Médecine, University of Bordeaux (Dr. Portmann); Prof. Dott., University of Milano (Dr. Bozzi), and Medical Academy Docent in Otolaryngology, University of Krakow (Dr. Szpunar).

Otosclerosis

Otosclerosis is the commonest cause of deafness in adult life. In 2,000 hard of hearing adults who attended a special clinic to be fitted with a hearing aid, no less than 944 (47%) were found to have otosclerosis. A review by Cawthorne of the clinical features of otosclerosis, based on records of 2,000 cases seen in the past 10 years, reveals that 10% had pink-tinged tympanic membranes. It is suggested that deficiency of cerumen and reduced sensitivity of the skin lining the external auditory meatus are the result of repeated otological examinations rather than an indication of altered vascularity as the result of otosclerosis. It is unlikely that the stapes is often immobilized by otosclerotic bone, though its movement is usually impeded. Therefore, it is suggested that stiffness of the stapes is a more accurate description than fixation or ankylosis, both of which imply immobility. Immobility is found only in very advanced cases where the oval window is totally overgrown by new bone. It is felt that the underlying cause of otosclerosis is an attempt by the body to change cartilage into bone. The site of election in the otosclerotic

process is the fissula ante fenestram, and this may contain an immature form of cartilage which is likely to be changed into bone at a time when bone growth-promoting substances are circulating in the body, as, for instance, during adolescence and in the later months of pregnancy. It is suggested that for a fenestration operation to be successful there must be a mobile window on each side of the basilar membrane. It is conceivable that the round window and its niche may be occluded by otosclerotic bone and the oval window left free. In such a case, a fenestration operation would result in two mobile windows on the vestibular side of the basilar membrane. The consequence of this would be a fluid wave which stimulates the vestibular mechanism without affecting to any great extent the cochlear mechanism. This may explain the cases in which a fenestration is followed by a drop in the hearing, a severe vertigo, and a very active fistula sign. The reason that the Tullio phenomenon does not occur after the fenestration operation is because the oval window does not readily move. In cases in which a fenestration operation has been done and there has been a mobile oval window, this usually results in an acoustic stimulus causing a vestibular response—in other words, a Tullio phenomenon. For this reason, a fenestration operation would be carried out only when the normal movement of the stapes is impeded. Closure of the fenestra after operation is not common, and in 850 operations that were done more than two years ago it has happened in only 8% of cases. In a further 13% of cases there was no improvement at all in hearing, though in nearly all of these an active fistula sign was retained.

Electrophysiological examinations in animal experiments (with the experimental type of otosclerosis) have shown a state of increased activity of the cerebral cortex, with absence of the labyrinthine oscillations and reaction to sound. It is assumed that, in patients with chronic impairment of the sound impression reaching the labyrinth and the cerebral cortex, there is initially a state of enhanced sensitivity of the auditory cerebral cortex, followed later by inhibition. The participation of the cerebral cortex in the mechanism of hearing is demonstrated by the deterioration of hearing in these patients in emotional states. The experiments which Khilov carried out on six rats, without decortication, demonstrated the development of the typical degenerative changes in the spiral ganglion and in the organ of Corti as well as the development of connective tissue in the scala tympani and the ductus cochlearis, under the influence of prolonged and strong stimulation. In six decorticated rats, the development of the degenerative changes in the spiral ganglion and in the organ of Corti was less obvious and there were no changes in the cochlea. It seems that for the development of the trophic changes in the labyrinth the functioning cerebral cortex is essential. It is concluded that otosclerosis is the sequel to disturbed trophic functions of the cerebral cortex. The clinical observations of Khilov have shown that the striking improvement of the hearing in the operated-on and in the opposite ear following fenestration of the labyrinth occurs in patients in whom there is predominance of the inhibitory processes in the auditory cortex before operation.

After discussing the genetic aspects of otosclerosis and the difficulties in interpreting genealogical trees in case histories, Nager points out that the difficulties of interpretion are due to the fact that otosclerosis is so often asymptomatic. Whereas otosclerosis may be found histologically in many parts of the temporal bone, it is only those otosclerotic areas occurring in the anterior margin of the oval window which produce hearing loss.

A study was made by Jengo and Vitto-Massei of the variations of the rhesus factor in otosclerotics, in two families with three generations each. The findings were as follows: 1. There is a definite parallelism between the two hereditary factors. 2. The

hereditary character studied is independent of the sex chromosome. 3. Both the rhesus-negative factor and the otosclerosis factor are of recessive nature.

In studying the metabolism of the bone in otosclerotics, Herrmann and Maurer found no pathology in the calcium and phosphor determination but the activity of the alkaline serum phosphatase was remarkably lowered. The cholesterin values were variable, as in normal persons. The lipoid phosphor determinations were normal throughout. The globulin-albumin fraction was normal. Otosclerosis was thought to be caused by inner secretory disturbance. C 17-ketosteroid excretion in the urine was studied. A lower level than normal was found in patients with otosclerosis.

Hlavacek, Opplt, and Musil described the electrophoretic spectrum of the blood serum proteins in uncomplicated otosclerosis. They found (in two-thirds of the cases) a characteristic increase of middle globulin fractions (α, β) most frequently jointly but also singly. For an interpretation of this pathologically changed proteogram they consider it essential to supplement the findings by an estimation of the lipoproteins and glycoproteins of the blood.

In 53 patients with otosclerosis, serum protein investigations were done by Vyslonzil. In 29 patients the β- or γ-globulin was increased and the albumin slightly decreased. In 25 patients sternal puncture was done. Ten times the number of myeloid plasma cells were at the upper normal border. Of 30 patients with otosclerosis, 28 had decreased prothrombin time and heparin tolerance. The chemical and cellular changes in the blood are explained as a sequela of a changed humoral equilibrium. The cause may be hormonal, changed vegetative reaction, or changed capillary fraction.

Otosclerosis is combined in 50% of the cases with inner ear damage. A large number of these cases turned out to be of autonomic origin (endo or perilymphatic hydrops). Moritz tried (1) to diagnose the autonomic factor of otosclerosis and (2) to obtain by a causal and symptomatic therapy (*a*) the proof of the autonomic factor, (*b*) to get practical therapeutical results. The diagnosis is based upon (1) lowered bone conduction and (2) other autonomic disturbances in the head-neck region (headaches, vertigo, globus, neuralgic disturbances, shoulder-arm disorders). In autonomic hearing disorders, the most frequent graph of bone conduction is slowly decreasing from the low to the high frequencies. The sudden drop at 2,000 frequencies is, in his experience, not of autonomic origin. Recruitment phenomenon, Ruge-Zangemeister (air + bone conduction with water-filled ear canal) and Gelle (air conduction with increased air pressure), are important for diagnosis of stapes fixation. Air conduction and bone conduction are identical in autonomic disorders but differ markedly in otosclerosis. Two cases are reported with cervical deformation, where the bone conduction (autonomic factor) could be improved by sympathetic blockage, extension, and manual treatment of the cervical spine and Phenotiazin sedation. The air conduction (stapes fixation) was not improved. Pseudo-otosclerosis is called a clinical otosclerosis combined with a severe inner ear conduction disturbance when both air and bone conduction are improved by the above-mentioned therapy. There is no stapes fixation; otherwise the air conduction would not improve. A subacute labyrinth hydrops is the probable cause of the inner ear disturbance. Fenestration would have been unsuccessful. In all otosclerotic patients, he first tries medical therapy. When air and bone conduction are improved and the two graphs approach each other (pseudo-otosclerosis), an operation is not indicated. In case only the bone conduction is improved, the stapes fixation is far advanced and fenestration is indicated. When air and bone conduction are not improved an irreversible degeneration of the organ of Corti is present.

A study was made by Maggio of the biological and clinical effects in subjects with otospongiosis of a treatment with

vitamin E and male sex hormone. The doses were adapted for every case but, on the whole, averaged 10 mg. of tocopherol acetate and 25 mg. of testosterone propionate per day in two cycles per year, each lasting 15-20 days. The conclusions reached can be summarized as follows: 1. Otospongiosis entails: (*a*) an increase of basal metabolism, (*b*) a decrease of the total urinary excretion of 17-ketosteroids, (*c*) a decrease of serum cholinesterase, (*d*) arterial hypotension, (*e*) a sympathicotonic tendency of the autonomous nervous system. 2. After the treatment, the phenomena mentioned under conclusion 1 were normalized. 3. The clinicoaudiological effects of the treatment described were lowering of the sound-perception threshold for air and bone conduction to an average value of 15 db., enlargement of the auditory field, functional restoration of completely inactive receptors, improvement of the intelligibility of speech and of subjective acoustic sensation, and disappearance or attenuation of tinnitus, as well as marked improvement of the general condition. As regards the duration of the results, it is stated that the auditory gain is not permanent but persists for periods of considerable length (even up to a year) and in any case, even if no prolonged improvement is obtained, at least the progressive deterioration of hearing is stopped. This can best be obtained by successive cycles of treatment. It is stated that the otospongiosis patient suffers from neurodystonia of sympathicotonic nature and from hyperfunction of pituitary and thyroid and that the auditory organ of these patients is also dystonic as regards the nervous regulation of the blood supply of the neurosensorial elements (angioneurosis of the branches of the spiral artery), resulting in vascular spasm in this region, weakening of the acoustic vasomotor reflex, and disturbed balance of the labyrinthine fluids, especially toward hypertension. In the osseous structures, the opposite phenomenon, capillary dilatation, occurs; it can be demonstrated indirectly by study of bone conduction before and after treatment with vitamin E and male hormone. The author considers the hypacusia of otospongiosis to be of purely functional nature and includes this form of otopathy among the "labyrinthoses."

The different methods regarding operative procedures in fenestration are discussed by Kroeth. Indications for fenestration are (1) benefit from even partial improvement of hearing power in a psychial and practical way and ability to apply a hearing aid more efficiently, (2) binaural improvements by improvement of hearing of the not operated on ear, (3) general improvement of hearing power by stimulation of the vestibular system, (4) influence on the hearing automatism by stimulation of the sympathetic system, (5) insecurity to forecast the improvement of hearing for all frequencies (especially to emphasize the possible utilization of the reversible inner ear component), (6) progression in otosclerosis often stopped by the operation.

An attempt is made by Femenic to contribute to the solution of several problems regarding noninflammatory labyrinthine affections, such as otosclerosis and Ménière's disease. The risk of closure of the surgically made labyrinthine aperture is avoided by the author with a procedure which he proposed for the first time in 1948 and which has since proved very effective. During opening of the new window, the endosteum should be preserved. This endosteum is incised with a sharp-pointed knife along the longitudinal axis of the newly formed window, and the two endosteal layers are placed on the osseous edge of the window and fixed in this position. In this way, any apposition of bone or connective tissue to the bony edge of the window is avoided. Shambaugh, in 1951, proved the efficacy of the method in a large case material. He termed such a window an "endosteumhemmed fenestra." In cases of labyrinthine hydrops refractory to treatment, the endolymphatic space is evacuated into the perilymphatic space. In this way, vertigo in three patients was completely

abolished and hearing in the operated on ear was preserved.

Antoli-Candela Cebrian has modified Lempert's and Shambaugh's surgical steps. He uses general anesthetic plus controlled hypotension. The mastoid approach has been reduced to an anterior atticotomy. His window is made stepwise; its facial border is left lower than the mastoid level. The tympanic flap is replaced by a dermic graft. The author claims better immediate and later results.

Suggit performs a closed one-stage fenestration as follows. The postaural incision follows closely the crease behind the auricle and turns forward above the auricle. The mastoid cells are removed down to the additus. The facial ridge and the bridge are removed, care being taken to separate the soft tissues of the meatus down to the tympanic ring. The incus is removed; the fenestra is made. The skin graft is then taken from the skin just posterior to the mastoid wound. A split-thickness graft, 1 cm. square, is shaped with a cataract knife. A little Ringer's solution is run into the cavity, and the skin graft is floated off the knife, applied over the fenestra, and molded into the fenestral opening. Two pieces of Calgitex gauze about 1 in. long are packed over the graft, and suture of the postaural incision is begun. When the incision is half closed, the two pieces of Calgitex are removed, and the graft is seen adhering to the bone surface. No packing is left inside. The suturing of the incision is completed, and a fine drainage tube is left within. The tube is removed in 24 hours. Suggit states that the results obtained with his technique compared favorably with the classical fenestration. The results of closed fenestrations refute the belief that it is necessary to separate the new fenestra from the tympanic cavity to obtain a gain in hearing.

At the otological clinic in Bern, the otosclerosis operation is done in two stages, by Escher. The first operation opens the middle ear postauricularly; then endaurally a flap is formed from the superior part of the external ear canal to cover the horizontal semicircular canal. Resection of the head of the malleus and of the incus is done. The window for the sound fistula is prepared at the ampullar part of the horizontal semicircular canal but not opened. The plastic flap is placed on the semicircular canal; the whole cavity is covered with Thiersch epidermis flaps. After complete healing (two months) the second postauricular incision is done. The horizontal semicircular canal is carefully exposed by lifting the plastic flap. In this restricted area the perilymphatic space is opened and the fistula covered with the plastic flap. The advantages of this second-stage operation are (1) minimal labyrinthic reaction and (2) minimal stimulation for osseus closure of the fistula. The result of hearing gain depends on the nerve function.

In the Zurich otorhinolaryngological clinic the fenestration operation is carried out in two sessions. The experiences obtained with this method in 400 patients are reported by von Schulthess; 255 were ideal or suitable surgical cases (according to Shambaugh). The result was very good in 277 cases (pure-tone audiogram better than 30 db.) and satisfactory in 96 (between 30 and 40 db.). There were 37 surgical failures due to an unknown cause. The improvement was persistent for at least two years in 76% of the patients operated on and for five years in 50%. Tinnitus was affected by the operation in only one-third of the patients. Three patients became deaf postoperatively.

The conditions to be observed for anesthesia during a fenestration operation are enumerated; these are, fundamentally, maximum quiet and perfect hemostasis. The latter is absolutely essential and must be perfect at the moment the fenestra is pierced. Twenty cases of fenestration carried out by García-Ibanez are analyzed by Vento and Torres. In these cases Arfonad was used as the hypotensor agent. The chief advantage of this drug is that it

is easy to administer and to control in comparison with other hypotensors.

Stirling reports the use of planned hypotension to reduce hemorrhage during the last phase of fenestration. Pentamethonium and hexamethonium were used to produce the hypotension. Electrocardiographic studies showed no disturbance of coronary circulation. Oximetric studies revealed adequate cerebral oxygen consumption. The author concludes that controlled hypotension is safe and helpful in fenestration surgery.

The problem of osteogenic closure of the fenestra has been largely solved. The chief cause of failure, report Shambaugh and Takahara, is now serous labyrinthitis. It is usual to observe an immediate improvement in hearing postoperatively, followed quickly by a depression. Thereafter there is in most cases an improvement, reaching a maximal level in a period of a few days to a few months. A classification based on 141 animal cases indicates that five grades of postoperative labyrinthitis may be recognized, varying from localized serous to diffuse purulent. A careful attempt is made to correlate histological findings in animals with observations made in clinical practice. Assuming that surgery is skillful, some degree of labyrinthine reaction is to be expected. Bleeding into the fenestra is probably the major cause of significant labyrinthitis, but infection and other factors as yet undefined must also be considered. Several surgical techniques have been used, and the influence of preoperative suitability has also been taken into account.

After fenestration the improvement of practical hearing is often attributable not only to the elevation of the threshold of hearing but also to a moderate recruitment and a lowering of the threshold of pain, according to Maspetoil and Semette. In a number of patients the authors observed a decrease of the difference-limen after fenestration. This "moderate recruitment" is caused by vasomotor alterations in the cochlea due to the operation and not by the absence of the protective mechanism of the stapedius muscle. In some cases the vasomotor alterations in the cochlea have a tendency to increase, with harmful effects upon the sensory cells. Thus the initial improvement of hearing may turn into an ultimate hearing loss.

It was shown by Skoog and Shambaugh that in many cases of fenestration hearing could be improved after the fenestration by loading the tympanic membrane with a swab soaked in paraffin oil or Aquaphor. Lempert believes that, if hearing is improved by loading the tympanic membrane with a swab, this proves that the fenestration has not led to optimal remobilization of the fluid of the inner ear. The optimal remobilization has been obtained, however, in cases where the swab does not lead to improvement of hearing.

Most authorities consider the hearing improvement after the fenestration operation permanent if it lasts for more than two years. Jauerneck examined 120 patients with otosclerosis who had undergone operation more than two years before and found that 90% had improved hearing. The same patients had been examined during the first two years after operation, when 95% of the results were found to be successful. The author does not think that a persistent discharge from the fenestration cavity is a cause of failure. He found occasionally that the hearing continued to improve and that it was better after two years than at shorter periods after operation. In patients examined after an interval of two years, 50% heard the conversational voice at a distance of 18 ft.

Salomin Danic deals with the causes of failures noted in the course of fenestrations executed during seven years and with the results of such interventions. Time is the best test for the value of this operation; therefore, only those fenestrations will be considered that date at least one year from the intervention and were effectively reexamined after one year. Two kinds of failures following fenestration can be dis-

tinguished. Primary failures are those which do not show any hearing improvement after the operation or in which deafness has even grown worse. Secondary failures are those where fenestration caused an auditive improvement, which later disappeared. Of 431 operated on, with a satisfactory function of the inner ear, 44 failures were noted; 20 of them were primary failures, including 2 aggravated deafness, and 24 were secondary ones. Of 75 operated on the limit of operableness, 10 were failures; 6 of them were primary, including 1 aggravated and 4 secondary. Of 26 operated with remarkably involved inner ear, 10 were failures; 9 of them were primary, including 2 aggravated and 1 secondary. Three reasons might explain primary failures: wrong operative directions, an excessively involved inner ear, and a serous labyrinthitis. In secondary failures, 29 operated on showed some hearing gain, which later disappeared. The closing of the window caused a failure in 28 of them. A threat of closing usually appears about the second or third month, with a decline in the auditive gain at the level of bass frequencies. During a more or less long time, improvement persists at 1,000 or 2,000 frequencies, letting the patient and the surgeon hope for a partial success of the intervention. Actually this decline remains limited in some cases to the bass zone, and the patient holds his auditive gain. This limited gain is often destined to disappear and usually takes hearing back to the preoperative level. The window closing became complete after 1 year in 19 patients and after 18 months in 7 of them. Two years may be considered sufficient to eliminate the risks of a closure due to osteogenetic process. Reports on such interventions followed by window closure show that in four cases osseous splinters persisted in the canal, which were not extracted for fear of injuring the membranous labyrinth. Ten operations were particularly hemorrhagic. In 10 operated-on patients the intervention seemed to be successful and there were no complications as to window closure. The percentage of window closure doubtless depends on the operative technique for most cases. A window which was executed according to the classical rules, under microscopic control, with well-prepared edges, and clear of all impurities, particularly of osseous splinters and blood, has every chance to be permanent. The results of fenestration depend mainly on the functional reserve of the inner ear.

1. *Patients Having Satisfactory Function of Inner Ear.*—Of 431 operated on, 395 (91%) hold an average gain of 26 db. after one year; 264 operated on (66%) recover their total hearing (i. e., an average lower to 30 db. in conversation zone); 111 (28%) partially recover (average between 30 and 40 db.); 20 hold their hearing gain, but their average in conversation zone still exceeds 40 db.; 302, showing good results one year after the intervention, were examined after two years, 296 of them still hold an average hearing gain of 26 db. Recovery was total in 203 of them (68%), partial in 75 (25%), none in 18. After three years, 172 improve 25 db.; 104 have a total recovery (60%); 54 (31%), a partial recovery; 14, no recovery. Forty-nine examined five to seven years after the intervention have an average recovery of 25 db. The recovery is total in 29 (59%), partial in 14 (28%), none in 6.

2. *Patients at the Limit of Operability.*—Of 75 patients, 66 (88%) hold an average gain of 25 db. after one year. Recovery was total in 9 (12%), partial in 45 (60%), none in 12. Forty-four showing hearing gain after one year were examined after two years; forty-three of them still had an average gain of 22 db. Recovery was total in 6 (13%), partial in 26 (60%), none in 11, and 12 examined five to seven years after the intervention have an average gain of 21 db. There was total recovery in 1 (12%), partial in 6 (60%), and none in 5.

3. *Patients with Bad Function of Inner Ear.*—Only 15 of 26 still hold an average auditive gain of 23 db. The recovery is partial in 5, none in the remaining 10. We can note after four to five years a slight

decrease in the average auditive gain, due in most cases to a slight deficit in the different frequencies and probably caused by the thickening of the edge. More rarely this deficit is due to a decreasing of the gain, involving only frequencies 2,048 and 2,096, and may be ascribed to an alteration in the inner ear.

After a brief review of the literature on the problem of maintaining a patent artificial fenestra in otosclerosis, Berlendis and Negri relate the results of their experimental research work on dogs. Small osseous cylinders were taken, by use of a special burr, from the palate, where canals of palatine vessels are to be found. These cylinders, which showed a small canal passing through their major axis, were grafted in the anterior part of the frontal sinus of the same animal. Microscopic observation could no more detect either the graft or the canal, starting from the first week after the intervention. Microscopic observation showed a quick reabsorption of the graft, which becomes entirely substituted by newly formed connective tissue, which becomes ossified later. The graft of an osseous cylinder with the central hole, far from hindering osteogenesis, stimulates proliferating activity of surrounding tissues, thus favoring the closure of the artificially made osseous injury. A practical application of such a method appears, therefore, to be absolutely irrational.

After enumerating the different techniques suggested for avoiding osteogenetic closure of the labyrinthine foramina in fenestration, deStefani, on the basis of previous experiments on the inhibiting action of ultrasounds, applied ultrasonic irradiation to labyrinthine fistulae produced on four dogs. He describes the surgical technique used and the histological method followed in studying the parts. Examinations were made on the 28th, 61st, 90th, and 124th days, respectively, following the operation, showing that there existed an inhibition and/or marked delay in the osteogenesis of the cases in question.

Halliday, Raffan, and Mackay use as criteria of a successful fenestration the fact that the air-conduction loss of 2,048 must not be greater than 30 db. and that on clinical examination the patient must hear well by both conversation and whispered-voice tests. They believe that skin grafting leaves the cavity cleaner and promotes earlier healing. Stenosis of the meatus was avoided by an adequate removal of the fibrous tissue overlying the temporal muscle at the upper end of the wound. The first dressing should be carried out with the same scrupulous regard to asepsis as the operation itself.

It was established by Femenic in patients who had been followed for more than five years after fenestration in otosclerosis that the improved hearing capacity had been persistent for this long period. A few patients showed insignificant fluctuations of the operated-on ear, which were about 10 db. in the intensity range of speech. However, the nontreated ear exhibited progressive deterioration of hearing and marked signs of degeneration of Corti's organ. In order to prevent, if possible, later closure of the newly formed window, the author proposed as early as 1948 first to preserve the endosteum in the making of the osseous aperture. This endosteum is then incised along its long axis, and the two endosteal plates thus formed are placed over the bony edge of the window. In this way, the wound edges of the window are covered with epithelium and regenerative processes of the bone and connective tissue are inhibited. In those cases in which a window could be formed in this way, secondary closure of the newly formed orifice is impossible.

Hutchinson has shown that better results are obtained when the fenestration operation is done for the chronic adhesive process compared with otosclerosis. As regards preoperative prognosis, it thus assumes importance to differentiate between the two types of impedance deafness. In addition to history, clinical examination, and audiometric

curves, x-rays can help to differentiate between the otosclerotic labyrinthine capsule and the normal found with the chronic adhesive process. Details of the modified Townes and Stenver's projections used are described, together with the points of value in differential diagnosis and x-rays for comparison.

Rosen and Bergman report their experience with mobilization of the stapes for deafness due to otosclerosis. The techniques reported 60 years ago in which the mobilization or stapes extraction was carried out through a myringotomy opening are reviewed. A new technique, in which a circumferential incision is made several millimeters lateral to the posterior half of the tympanic ring, is devised. The skin deep in this incision is elevated and the adjoining tympanic membrane freed from its insertion into the sulcus. The freed drum is reflected anteriorly. If further exposure of the incudostapedial articulation is needed, then a little of the overlying bone is removed with a fine curette. The stapes is mobilized by applying pressure to the anterior aspect of its neck in the direction of the stapedius muscle.

Improved hearing after mobilization of the stapes in otosclerotic deafness is reported by Rosen and Bergman. The following modification of Kessel's operation should first be practiced extensively on the cadaver and a special set of instruments employed. A tympanomeatal flap is elevated. The notch of Rivinus is nibbled away so as to expose the stapes more completely. Normally, the tendon of the stapedius vibrates if the ossicles are touched. An ankylosed stapes can be mobilized by pressure in the axis of the stapedial tendon, without fracturing the crura. It yields with an audible snap. The advantage over fenestration (if that operation has not already been performed when it may still be beneficial) is that physiological ossicular conduction is restored.

Rosen, in 1952, described a technique of mobilization of the stapes which he now refers to as the indirect method. In this technique, pressure is exerted posteriorly against the anterior aspect of the neck of the stapes until the fixed footplate is broken loose from its bed. The direct method is one of prying loose the footplate at one, two, or three places at the footplate itself. The exposure technique is the same as that in the earlier operation, except that it is now essential in all cases to remove 2 to 3 mm. of the edge of the posterior bony canal wall. This permits a full view of the incus, long process, incudostapedial junction, etc. At first the indirect method of mobilization is tried. If this fails the direct method is applied. In this way it is possible to achieve success in the types of situations that were failures by the indirect method.

A preliminary report, reviewing the results of 50 cases of clinical otosclerosis treated surgically by transtympanic mobilization of the stapes, is presented by Scheer. The cases were operated on according to the Rosen method. The fact that this method can restore hearing in otosclerosis is established. The stapedes were found fixed in 94%; in 62% they were mobilized, but only 30% got a return of hearing to a serviceable level. In 16 cases (32%) the stapedes were fractured because they were too fixed. Histories and audiograms of the successful cases were presented. Two stapedes with successful mobilization became refixed in the oval window and were remobilized, with a return of hearing to the original postoperative level. Fenestration can be successfully performed after an unsuccessful mobilization. In 10 cases there was a sacrifice of the chorda tympani. In all these patients disturbances of taste were observed, which seemed to remain to a slight degree.

Scheer reports a series of 50 cases of otosclerosis treated by stapes mobilization. He obtained 30% of satisfactory results where the hearing improvement reached or surpassed the 30 db. level in the speech frequencies. Of these successful cases 86% have maintained this gain for one year. Rosen, in discussing this paper, stated that the selection of cases and the description

of results in mobilization must be based upon careful and complete audiologic evaluation, including pure-tone air and bone and speech audiometry. Such details as calibration of equipment, selection and calibration of proper masking for the bone-conduction tests, control of test conditions, and skillful testing, preferably by a third party, will do much to validate this as an important procedure for the relief of deafness due to otosclerosis.

Stapes mobilization is not a reasonable substitute for fenestration surgery, as its application is not sufficiently effective in those cases which are ideally suited to fenestration surgery, reports Kos. Stapes mobilization may be useful in patients exhibiting beginning or incomplete ankylosis and in patients whose bone-conduction thresholds are elevated to an extent which precludes a successful fenestration operation. Surgical mobilization of the stapes is still experimental in the sense that the likelihood of lasting results remains purely speculative.

After 65 transtympanic stapedial mobilization operations, Kos has learned a number of pertinent facts and has gained some interesting impressions. The technique is satisfactory, but it is a hit-or-miss procedure, with no detailed criteria evident for selection. Predictions cannot yet be made with a significant degree of confidence. According to the author, three categories of clinical otosclerosis must be considered for the stapedial mobilization procedure: (1) incomplete stapedial ankylosis, (2) complete stapedial ankylosis, and (3) stapedial ankylosis with inner ear hearing deterioration. The first two categories are based on normal bone-conduction thresholds for pure-tone audiometry. While regression of hearing improvement may occur in Groups 1 and 2, it is found more frequently in the cases of beginning ankylosis. Usually it occurs four to six weeks postoperatively, but in the series at hand has occurred as late as six months after operation. The operation is most effective in the third group, where bone-conduction thresholds are so elevated as to preclude the possibility of a successful fenestration operation. In such cases, the hearing may be restored to the level of the bone-conduction threshold. It is concluded that whether stapedial mobilization will be retained in the surgical repertory of the otologist will depend upon future experience with the procedure and the long-term results to be evaluated in the next several years.

Stapes mobilization is most successful in niche otosclerosis with progressive impairment unilaterally. Bilateral otosclerosis with 40 db. loss and normal bone conduction will return to normal. In otosclerosis with 60 db. loss and reduced bone conduction mobilization is difficult, since the crura break easily before the foot plate gives way. Advantages of stapes mobilization, according to Zangemeister, are as follows: 1. It may be done on an outpatient basis. 2. There is no labyrinthic reaction, vestibular disturbance, intercurrent hearing loss, or loss in the higher frequencies. 3. There is normal middle ear, with no danger of infection. 4. Often there is full recovery of hearing as far as the inner ear is functioning. 5. There remains the possibility of later fenestration.

Meurman and Meurman report their experience with Rosen's method of stapes mobilization in otosclerosis. Improvement in hearing was obtained in 37 (58.7%) of 63 patients operated on. An improvement of 20 db. was taken as the borderline. Since the time of observation was short, no permanent results were reported. Accidental complications at the operation, such as luxation of incudostapedial joint, rupture in pars tensa, and facial palsy, may occur. The chief cause of failures is fracture of one or, occasionally, both of the crura.

Restricted visuality with Rosen's operation gave rise to a search for a method where better visuality of the stapes is granted. The opening of the attic is the method of choice, reports Meurman. Since experience shows that fracture of the stapedial crura happens in one-third of the cases, it is advisable to loosen the ossified

annulus. The atticotomy allows one to perform immediately a fenestration in case fracture of the crus of the stapes has happened. Use of the Zeiss microscope, with magnification 10-16, facilitates recognition of otosclerotic areas of the crura.

Daly and others describe the technique of mobilization of the middle ear structures by the passage of bougies into the middle ear cavity via the Eustachian tube. Cases are described where improvement in hearing following the procedure lasted two months.

Ménière's Disease

Lindsay reviews the problem of Ménierè's disease. Diagnosis is based on the type of hearing loss, the nature of the tinnitus, and other auditory disturbances. Occasionally in the earlier years of the disease the hearing loss primarily affects the threshold for low tones. The degree of hearing loss fluctuates and can be demonstrated on repeated audiograms. Tinnitus is of the buzzing or roaring type as distinguished from the high-pitched hissing tinnitus that goes with high-tone deafness. The intensity of the tinnitus varies directly with the fluctuations in the hearing threshold. A sense of "blockage" in the ear and sometimes of pain in the same area is also characteristic and varies in intensity with the hearing loss and tinnitus. The affected ear is sensitive to loud sounds, even though there is a marked hearing loss. Diplacusis is frequent. A characteristic of Ménière's disease is that, as the intensity of sound is increased above the threshold, the loudness as sensed by the patient increases more rapidly than in a normal ear (recruitment); hence the patient's ability to understand speech does not improve as the speech is made louder. This factor interferes with the benefit to be obtained from a hearing aid. With the presence of the above symptoms, a diagnosis of Ménière's disease can be made without the complaint of vertigo. Clinical observations have indicated that any measures that can be taken to reduce nervous tension and promote regular habits of work, rest, and eating appear to have a beneficial effect on the disease. In severe, stubborn, and disabling cases consideration of surgical treatment is justifiable. Operation is aimed only at the prevention of dizziness.

Perceptive deafness is characterized by impairment of high tones. Nakamura states that instances of perceptive deafness with low tones may be found in cases of Ménière's disease. The cause of this type of deafness has been explored in relation to Ménière's disease. Williams regarded it as a kind of "endolymphatic hydrops without vertigo." The existing theories, however, are not able to explain satisfactorily the development of hearing impairment in these cases. An experimental study was made to discover the cause of such perceptive deafness with gradual low-tone loss. The author observed the development of gradual low-tone loss in air and bone conduction when auditory fatigue was caused by such noise as filtrated by a pass filter of 200-400 cps. This type of deafness may have its cause in the peripheral neural elements of the auditory organ.

On the basis of 46 personal observations of labyrinthine vertigo, Ivanovic is inclined to admit Mayoux's conception that the examination of the fundus oculi and of the pressure of the blood vessels of the fundus oculi allow the possibility of an adequate treatment of Ménière's disease, supposing, of course, that similar changes are taking place in the blood vessels of the labyrinth. The neurovascular disturbances of the basilar cerebral arteries certainly play a great part, perhaps even a decisive role in the appearance of peripheral vertigo. By the repeated examination of the fundus oculi and the evaluation of the pressure of the central artery of the retina we are in a position to track down early the vasomotor disturbances of the labyrinth, even if there are very slight changes in the inner ear. Thus, by appropriate treatment we are able to prevent the appearance of the clinical picture of Ménière's syndrome.

Good results have been obtained in the treatment of Ménière's disease by Podvinec,

using Mayoux's principles of testing the pressure of the central artery of the retina. It was supposed that among patients with perceptive deafness of unknown origin there might be cases in which hypertension or hypotension of the labyrinthine fluids causes damage to cochlear function without producing obvious vestibular symptoms. In some cases of perceptive deafness a marked difference existed between the blood pressure in the brachial and the retinal artery. After a series of intravenous injections of hypertonic or hypotonic solution, in most such cases a marked improvement of hearing could be noted.

Lindsay and Siedentop review treatment of Ménière's disease at the University of Chicago clinics from 1940 to 1954. Surgical treatment was considered necessary in less than 10% of the cases. In the authors' experience, operative procedures on the labyrinth attempting to preserve hearing and destroy vestibular function have resulted in recurrences of vertigo, frequent failure to relieve tinnitus, and consistent failure to preserve hearing. They, therefore, consider that exenteration of the ampullae of the horizontal and superior semicircular canals with the contents of the vestibule is the most satisfactory treatment.

The principle of ultrasonic treatment to the labyrinth, according to Arslan, is to eliminate the vestibular function in severe Ménière's disease without damage to the cochlear nerve. Improvement of dizziness took place immediately or after some months. Improvement of hearing and tinnitus occurred as well in 40%-50%. The effect of the ultrasound is threefold: (1) thermic, (2) mechanic-vibratory, and (3) chemical-physical. The third is a sequela of the others. The "cavitation phenomenon" takes place owing to release of gases in the living cells. Two hypotheses explain the successful treatment in Ménière's disease: (1) destruction of neuroepithelium of the crista and (2) effect on the endolymphatic hydrops, decreasing hypersecretion of the endolymph or facilitating its resorption. The present use of ultrasound externally is not effective, since the air in the mastoid cells and middle ear prevents conduction of the therapeutic doses. Direct application to the osseus labyrinth is done in two ways. The different steps are (1) antrotomy under local anesthesia; (2) exposure of the lateral semicircular canal; (3) ultrasound radiator brought in contact with convexity of lateral semicircular canal with the best possible direct contact and with the direction of the radiation avoiding the facial nerve or the cochlea; (4) continuous observation of the nystagmus imperative, with the nystagmus starting one-half to one minute after beginning of the radiation, first of irritative type toward the radiated ear, after some minutes to the other side as a paralytic nystagmus; (5) careful control of bleeding. Endaural application is only possible, where fenestration, atticoantrotomy or tympanoplasty was done before. Length of radiation is determined by the change in nystagmus direction, (25-30 minutes). The following cases were operated upon: 88 hydropic labyrinthoses (Ménière), 23 chronic adhesive hyperplastic otitis media with secondary labyrinthosis, 14 postotitic labyrinthoses, 32 otosclerosis to improve the hydrodynamics of endo- and perilymph and to prevent osseus regeneration of the fenestra "novovalis." Tinnitus was improved in most of patients. The postoperative course shows less severe reaction compared with the intercranial section of the vestibularis or after alcohol injection into the labyrinth. The vertigo ceased in 95%. Of 88 patients, 4 had to have a second ultrasound application. Only once was the ultrasound application done bilaterally. The hard of hearing were improved markedly in the low and middle frequencies. Vyslonzil stated that, besides thermic effects, special unknown ultrasound effects are of importance. Morphologically they found hyperemia, hematoma, hydrops, and increased connective tissue in the labyrinth.

Patients were treated with ultrasound, by Naumann, for tinnitus, labyrinthine atrophy, and otosclerosis. No improvement in any of these conditions was noted. In several

patients, vertigo, changes in the drum membrane, and exudate in the middle ear were noted. In view of these reactions, ultrasound was used experimentally on the ears of animals. Histological studies on these animals revealed destructive changes in the temporal bone structures and the adjacent brain tissue. The conclusion is that one should be very cautious in applying ultrasonics for the treatment of ear disease.

Della Vedova wishes to prove that labyrinthine vertigo treated medically without success might be corrected by surgery. The patient will regain his static and dynamic behavior but should be carefully examined before any treatment is begun, since vertigo is not peculiar to labyrinthine pathology. Two methods can alter the function of the labyrinth: (*a*) Decompression of the labyrinth would reestablish or make easier the absorption of the excessive labyrinthine fluid, so that the hydraulic ram stroke and consequent vertigo might be avoided. (*b*) The vestibulo-occular disturbances would be suppressed by neurotomy of the eighth pair, even when limited to the neurotomy of the vestibular roots, or by labyrinthotomy. A few drops of absolute alcohol are to be injected into the labyrinth, according to Mollison, to be sure of its complete destruction. Neurotomy is not to be practiced in patients with good auditory function, but it is advisable in those patients whose vestibular syndrome is not clear and who might be suspected of having a lesion of the pontocerebellar angle. If the angle is normal, a total neurotomy is advisable. Alcoholization does not abolish *ipso facto* the little cochlear function still existing in the patient.

Critical remarks on the various operations devised for the treatment of Ménière's disease are made by Schlander. The most reliable operations are (1) intracranial section of the vestibular nerve and (2) the direct or indirect destruction of the labyrinth through a fistula in the horizontal semicircular canal. All other methods are uncertain owing to hypothetic suppositions or difficulties in control and are often complicated by incomplete vestibular destruction or facial paralysis. The operations should be reserved for unilateral severe Ménière's disease after conservative therapy has completely failed.

Yakushiji made a histological study of the inner ear after Lempert's operation was performed on cats and his findings were as follows: 1. After one week the change was limited to disappearance of the outer hair cells from the third turn of Corti's organ. The other structures in the cochlea remained the same as in normal animals. 2. Two weeks after, the outer hair cells of the first half of the first turn and the whole of the third turn were found to have disappeared. 3. Four weeks later the scala tympani near the round window was filled with fibroblastic proliferations, with calcification in places, and the round and oval windows were already repaired. The outer hair cells and Deiter's cells of the beginning of the first turn were lost. In the other part of the turns, however, Deiter's cells were retained, although the outer hair cells were almost lost. 4. No changes were found in the sensory cell of the vestibule.

Patients with Ménière's disease are usually extremely tense and nervous and hence need reassurance. The definitive treatment can be divided into that for the acute attack and that for the persistent underlying condition. For the former, Williams describes the use of atropine, epinephrine, amphetamine, and dimenhydrinate (Dramamine). For the latter he recommends a low-sodium, acid, or neutral ash diet, with the administration of ammonium chloride. It has been found more effective to combine several methods of treatment: vasodilators to unlock arteriolar spasm, the acid ash low-sodium diet to decrease endolymphatic hypertension, endocrine therapy when indicated, and sedatives to relieve tension and anxiety.

Disorders of the general fluid balance, disorders of the blood circulation in the inner ear, and nerve disorders concerning the smallest blood vessels are responsible for

hydrops of the labyrinth. Surgical treatment and ultrasound therapy show good results in Ménière's disease. Conservative therapy is done with estrogenic and androgenic hormones. Best effect was seen with depot injections one time weekly of 65 mg. of testosterone plus 4 mg. of estradiol valerate, for three weeks. Eight days after the last injection 4 mg. of methylestosterone plus 0.002 mg. of ethinyl estradiol as tablets were given q. d. (Primodian tablets, Schering). Results were as follows: In 11 women over 40 years of age, there were no attacks in 6, attenuation in 4, and no effect in 1. In nine women under 40, there were no attacks in four, attenuation in two, and no effect in three. In four men: there were no attacks in three, attenuation in none and, no effect in one. Surprisingly good are the results in men, all of whom were above 50. The sex hormones affect the hypophysis especially by suppression of the anterior hypophysis, which in turn checks the adrenal and thyroid glands. With the mixed hormonal therapy, Bablik obtained peripherally an incretoric activation similar to the effect of cortisone and corticotropin (ACTH) but less dangerous.

Favorable therapeutic results in 47 cases of labyrinthine hydrops were obtained by Schenk by iontophoresis with carbaminoyl chloride. The active electrode was placed in the external meatus. Besides the specific action of the drug used, the beneficial effect is also ascribed to physicochemical alterations brought about by the electric current.

The diuretic acetazolamide (Diamox) given in labyrinth hydrops had the following results, as reported by Pruessing: 1. Nearly all patients treated were relieved of symptoms within three to four weeks. 2. The hypersensibility of the nervus vestibularis showed improvement more readily and returned to normal in a shorter time than the hyposensibility. The latter needed prolonged therapy. 3. The impairment of hearing was improved in most of the cases. 4. Serious side-effects or drug sensitivity were not encountered. The most useful dose was ½ tablet b. i. d. for four weeks (125 mg. to 250 mg.); ½ tablet every morning for two weeks and 2 tablets weekly for two weeks. Fluid was restricted to 1,000 cc. daily for eight weeks.

Therapy of motion sickness and Ménière's disease with a concentrated solution of sodium bicarbonate is reported by Hasegawa. Healthy rabbits rocked 63 times in one minute show a sympathicotonic condition (elevation of blood pressure and suppression of intestinal function). Rabbits centrifuged rapidly in order to detach the otolith were immune against motion sickness. Administration of 5% solution of sodium bicarbonate (5 cc. I. V.) dissolved the otolith (histologically proved), and motion sickness did not occur. Then experimentally 72 patients with remarkable motion sickness were treated with 5% solution of sodium bicarbonate (50 cc. I. V.). Fifty-five patients were cured; seven improved, and ten did not respond. Fabio Giaccai and Aldo Carle found in pigeons treated with sodium bicarbonate the crystals of the otolith still present in the labyrinths. The author believes that the crystals are not destroyed but that the organic membranes enclosing the crystals are. Untreated rabbits showed marked degeneration of the nervus vestibularis when put on a seesaw for a prolonged time, and frequently rabbits pretreated did not have any degeneration of the nerve ganglia. Prophylactic treatment of drivers may be considered. A total of 42 patients with Ménière's disease were treated. Thirty-six showed cure or marked improvement; six did not respond. As explanation, (1) the organic membranes are dissolved, which prevents exaggeration of the vertigo, and (2) the spasm present in Ménière's disease causing the edema is released by sodium bicarbonate (animal experiment). The chemical action of sodium bicarbonate is a transient lowering of the albumin-globulin ratio, of the viscosity and dilatation of the peripheral blood vessels. In man, 50 cc. of 5% solution of sodium bicarbonate is used. The injection

has to be done slowly over two minutes. Bad side-effects were not seen.

The following groups of patients were considered by R. Hahn in studying behavior of histaminic and nicotinic acid skin tests: (1) Ménière's syndrome by initial endolymphatic dropsy (vestibular reflexes normal with slight deafness); (2) Ménière's syndrome by endolymphatic dropsy with thermic and rotatory hyporeflexia, normal threshold of galvanic nystagmus, cochlear hypacusis; (3) Ménière's syndrome and neurotic evolution (thermic, rotatory, and galvanic hyporeflexia, hypacusis with recruitment); (4) Ménière's syndrome with a prevailingly vestibular onset or evolution; (5) Ménière's syndrome with a prevailingly cochlear onset or evolution. The results were summed up in the following observations. Intradermal reaction to histaminic acid is positive in most subjects suffering of initial Ménière's syndrome or of endolymphatic dropsy with cochleovestibular affection (in 3 cases of 5 and 4 of 16). Furthermore, the skin test to nicotinic acid is mainly positive in subjects showing an initial Ménière's disease (4 cases of 4) and less frequently in cases of endolymphatic dropsy with cochleovestibular affection (6 cases of 10). Finally, most patients suffering from Ménière's disease having a neurotic evolution show negativity (6 cases of 8) to histaminic and nicotinic acid tests. No difference is showed with regard to the behavior of skin reaction to histaminic and nicotinic acid among the cases of endolymphatic dropsy having a prevailingly cochlear expression and the cases of prevailingly vestibular expression. The data concerning the different forms of Ménière's syndrome can be quoted as a confirmation of the previous considerations regarding the difference in the results of intradermal reaction to histaminic and nicotinic acid in angioneurosis and endolymphatic dropsy. A high percentage of positive results to histaminic acid is present, with no particular relation to the symptoms showed by the different subjects; this figure is probably to be considered as the expression of an abnormal reactivity of the inner ear structures. Finally, negative results to both histaminic and nicotinic acid in Ménière's syndrome having a neurotic evolution show that either the etiopathogenetic mechanism is different from the dropsy or angioneurosis ones or the next neuritic evolution at an endolymphatic dropsy phase is no longer connected to any modifications in the subject's reactivity and in the vascular motility.

In a patient with a coronaritis and an arteritis of the inferior limbs, a bilateral Ménière's disease has been controlled, with good results, by an injection of cocaine in the middle ear on one side only, report Mayoux, Lafon, and Lesne. The ear signs disappeared on both sides, at least for a time. The authors emphasize the importance of such an injection. It shows the degree of permeability of the fenestra rotunda cochlea, the localization of a tinnitus, and the behavior, in certain circumstances, of the semicircular canals; finally, the possibility of an experimental action on the "interdependence" of ears is mentioned.

In a case reported by Leoni, where the cutaneous manifestations of Schönlein's disease were associated with Ménière's syndrome, both conditions showed a transient regression during the whole pregnancy period. The common denominator of both manifestations, cutaneous and labyrinthine, is thought to be an allergic condition determined by the sex hormones.

This is a preliminary contribution by Rius concerning iontophoresis for hydrops of the labyrinth and the vascular functions of the internal ear. This work is a further modification of similar reports on both the intratympanic action of various solutions and the effect of ionization on the internal ear. The author injects tolazoline hydrochloride (Priscol) supplemented by the hyaluronidase (Wydase). Tolazoline acts as a vasodilator, while the hyaluronidase facilitates diffusion of tolazoline into the internal ear, improves the local nutrition, improves any blood stagnation about the capillaries, and influences the endolymph and the hydrops. The drum is anesthetized with

topical 10% cocaine hydrochloride in pure aniline oil. After the anesthetic, he injects 0.1 cc. of tolazoline and 0.1 cc. of a fresh solution of hyaluronidase intratympanically. He inserts a specially constructed ear speculum into the external canal and fills the canal with a cotton pledget saturated with 12 drops of tolazoline. The positive pole of the galvanic current is connected to this ear speculum and the negative pole to any other area. The current is turned on to 1 ma. The initial duration is 10 minutes, but this can be increased to 15 minutes and finally to 30 minutes. Sometimes he may even increase the quantity of tolazoline to 0.2 cc. intratympanically and may also supplement the procedure by an oral administration of tolazoline on the day of the treatment. The author presents a series of 14 patients with rather interesting results. There were three cases of typical Ménière's disease in which the deafness, tinnitus, and vertigo all improved. There were seven cases of hydrops with or without initial vertigo; five had a definite improvement in hearing acuity, and in six there was marked improvement in tinnitus. There were four cases of Ménière's syndrome without hearing defects; three of the four presented improvement both in the tinnitus and in the vertigo. In Ménière's disease, in hydrops, and in Ménière's syndrome without deafness favorable results have been obtained. These results are definitely better audiometrically than those obtained by other therapeutic measures. Iontophoresis can be used as an important therapeutic test in the differentiation of Ménière's syndrome without hydrops from the vertiginous syndrome of other pathologic origins, such as neurologic and otitic infections, tumors, trauma, allergy, and toxic or degenerative processes. If time will prove the good results obtained by iontophoresis in controlling the vertigo of Ménière's disease, there will be no justification for medical treatment (streptomycin sulfate) or for surgical destruction of the internal ear (alcohol, coagulation, ultrasonic stimulation, and various operations on the membranous canal), which would sacrifice the hearing and yet not guarantee any results to the patient for the other ear.

Reconstructive Surgery for Deafness

Indications for surgery in hearing disorders are discussed by Majer. In severe bilateral otosclerosis with some nerve function a fenestration is indicated to obtain a better result from wearing a hearing aid. He advocates a hearing test with a probe, which is in apposition with the drum or crus brevis and attached to an ear phone for bone conduction. In stapes fixation the graph is 10-30 db. lower than the bone conduction. In normal ears or chronic otitis with small perforations, the graph is 20 db. higher than the bone conduction. Even with an interrupted ossicular chain, scarred drum, and mobile stapes the graph shows better results than the bone conduction. Stapes mobilization is performed (1) in beginning otosclerosis, since the gain in hearing is about 15 db., which is enough for sufficient hearing; (2) in patients with a hearing loss for the higher frequencies, and (3) in patients with occlusion of the window following fenestration. Tympanoplasty improves hearing in chronic otitis with loss of the drum and the ossicular chain. The aim of tympanoplasty is to restore a functioning middle ear with natural sound transmission. A positive prosthesis phenomenon with improvement of hearing identifies a well-functioning ossicular chain and vibrating windows. Large perforations of the drum closed by an artificial drum may show improvement of hearing only when the niche of the round window is filled. It can be assumed then that the chain is interrupted. In all patients with a positive prosthesis phenomenon, plastic operations of the middle ear are indicated. Closure of the perforated drum by a free graft or rotated flap after mastoidectomy will improve the hearing. A good function of the inner ear and a patent Eustachian tube are imperative. Hearing tests done by a probe inserted to the drum, crus brevis, stapes, and promontorium, com-

paring them with the bone conduction, are used. With an intact ossicular chain the graph obtained from the drum, crus brevis, and stapes is 25 db. higher than the bone-conduction graph. With an interrupted chain the graph is little different from the bone conduction but is 30-40 db. higher when taken from the stapes. In this way, vibration of the oval window is proven. In case the graph of the bone conduction is similar to that of the promontory and the oval window, it proves blockage of the oval window. Eustachian salpingitis calls for treatment before the operation. Scars may be removed surgically. Complete occlusion of the Eustachian tube is a contraindication of plastic operations in the middle ear. If possible, plastic surgery should be done with dry perforations. In case of a chronic suppuration of the mucosa and polyp formation the tympanoplastic may be done in two steps. Improvement of hearing may be achieved by transposing the incus against the stapes or forming a flap to cover the stapes. In bilateral microtia and atresia of the ear canal an operation is indicated in case the inner ear is functioning well and the child at least 6 years of age.

Malformation of the meatus and the middle and internal ear are dealt with and knowledge of the embryology is stressed by McAskile and Sullivan. Surgery is advised only for restoration of hearing and preferably as soon as good cochlear function can be assessed—about the age of 4 years. Operation should not be undertaken for cosmetic reasons; an auricle prosthesis is recommended. The operation must be adapted to suit the conditions present. The authors secured best results by removing the incus and malleus and placing a skin graft in contact with a mobile stapes. Where stapedial fixation is present the fenestration site is exposed and the whole cavity skin grafted. A fenestration is performed at a later stage after the cavity has fully epithelialized. A method of using skin grafts to secure a patent meatus without prosthesis is advocated. Eight cases are described and two in which there was congenital absence of the stapes.

Miyajima reported two cases of congenital atresia of the external auditory meatus combined with malformation of the auricle which were successfully treated by Scheibe-Pattee's operation. After this procedure their hearing ability was improved by some 20 to 30 db. in all frequencies tested.

In severe aural deformities no middle ear is present, according to Ungerecht. To find the inner ear is extremely difficult. The facial nerve or the labyrinth may be damaged easily. Aural deformity is often associated with hemiatrophia faciei or dysostosis craniofacialis. Owing to hypoplasia of the temporal bone, jaw, zygomatic bone, or petrosal bone, orientation is difficult. The rudimental auricles are displaced forward. Instead of an external canal a slightly depressed area is present. During operation lead marks are inserted into the wound and x-rays are taken to determine the distance to the semicircular canals by planography. In case the special x-ray apparatus is not available, the following operation is recommended. The hypoplastic mastoid or temporal bone is opened at the level of the normal external ear canal on the other side. The spongiosa of the petrosal bone is removed completely, the upper and posterior plane of the petrosal bone being exposed. Since the plane of the petrosal bone declines markedly lateral of the labyrinth block, the dura has to be exposed and lifted in order to reach the labyrinth block. With this method the posterior vertical semicircular canal is reached and opened. The opening of the horizontal semicircular canal is not recommended, since damage of the facial nerve easily occurs. The rudimental auricle is removed completely. The asymmetry of the skull is improved by fat plasty. An auricular prosthesis is later formed with a glycerin-gelatine material. Fenestration in these patients is done only when the hearing of the other ear is markedly reduced.

Altmann reports on the clinical and morbid anatomical findings of an infant born with multiple bone malformations, particularly in structures originating from the regions of the first and second branchial arches on each side. It is pointed out that stapes ankylosis was present on both sides although from different causes and hence surgical reconstruction which did not include fenestration of the bony labyrinth would have been inadequate. He also gives an account of the morbid anatomical findings in the ears of five rats, all of whom had unilateral congenital aural malformations. It is pointed out that, although different in degree, these malformations are basically similar to those found in the human. In each case, the mothers of the malformed rats had had injections of trypan blue before or during pregnancy. The malformations are attributed to this. In two of the rats, the malformations in the inner ear were identical with those in the offspring of male mice treated with x-ray. Comparison with a human temporal bone with bilateral atresia of the external auditory meatus has shown the same findings, although in rats the distal portion of the first branchial pouch and the derivatives of the upper part of the second branchial arch seemed more susceptible to malformations than in man.

It is emphasized that some congenital malformations of the ear exist other than the typical forms of auricular dysplasia, in which the bone barrier determines the atresia of the external acoustic duct, causing the complete acoustic isolation of the internal ear and thus justifying the traditionally accepted name of "atresia auri congenita." In other malformations there is an acoustic "deficit" conditioned by the inefficiency of the ossicles due to a malformation of one of its components, while the external acoustic duct is completely normal. Those malformations are called "small auricular dysplasias." This name not only indicates a less serious malformation but is also a differentiation from a practical point of view. Therefore the name "small dysplasy" has a diagnostic and surgical value, for in these cases the surgical therapy is different, as are the therapeutic possibilities. Bozzi, Cova, and Invernizzi state that only surgical exploration allowed the physician to discover such forms, the existence of which had already been suspected on the basis of the acumetric and radiological tests. The reported cases (three cases, five ears) demonstrate the variability of such malformations and of the results which may be obtained by using surgical techniques.

The average tympanoplasty case does not correspond to theoretical requirements, reports Wullstein. This is attributable partly to problems connected with the function of the two windows and partly to the ventilation of the newly formed tympanum, i. e., the mucosa of the tympanic cavity, and particularly the quality of the new tympanic membrane as a sonic pressure transformer and protector. It should be borne in mind that, in compromising with these two functions, even a normal drum does not always provide ideal sound protection for the round window, as shown by a modified technique of fenestration operation by which the entire structure of the middle ear apparatus is controlled but in no way altered.

The following methods of surgical restoration of the middle ear and its functions are distinguished: fenestration, stapes mobilization, tympanoplasty, and operative treatment of atresia auris congenita. The purpose of all these operations is to make conduction of sound from without to the perceptory organ and the hydrodynamics of the cochlea possible. To this end, the following procedures are necessary: (1) partial or complete reconstruction of the tympanic membrane; (2) new formation of an air-containing tympanic space, lined with mucous membrane, without epidermis; (3) preservation of the chain of ossicles as far as possible; (4) grafting of a makeshift mucous membrane, if necessary; (5) construction of a well-functioning sound protection for the round window, since with faultless functioning of the windows, the hearing loss is reduced to at least 25 to 30

db. In cases of stapedial ankylosis with fenestration in the lateral semicircular canal, the round window is in the sound protection of the preserved tympanic membrane and the chain of ossicles is eliminated as a transformer of sound pressure. When the tympanic membrane acts as a fully functioning sound pressure transformer, the hearing loss is decreased by another 25 db. Since the malleus and incus constitute a further gain of hearing by only 2.5 db., their functional loss is of no significance. In this case, the tympanic membrane which has been preserved or replaced by a skin graft should be placed directly on the stapes or an artificial stapes of synthetic substance, as in a columella system. The operative technique is described in detail by Wullstein. Not all the theoretical requirements can be met in every case partially owing to unavoidable cicatrizations of the windows, partially also to the ventilation of the new tympanum from the tuba, and further to the quality of the tympanic mucosa or its substitute and the quality of the new tympanic membrane as a transformer and protector of the sound pressure. On the above-mentioned grounds, the middle ear is not capable of ideally fulfilling its task in many cases. Hearing results with less than 15 db. middle ear components in the principal range of speech are therefore to be regarded as good.

In his paper Zöllner deals with the preservation and restoration of hearing function following large eardrum perforations and the other destructive processes accompanying suppuration or cholesteatoma of the middle ear. He stresses that the number of combinations of deformities that result from destructive disease are many and the total number of functional plastic repairs performed as yet are few; therefore, definite statistical conclusions of results are not of value. His basic discussion deals with free skin grafts in the middle ear and the problems encountered in their successful use from a functional standpoint. He mentions that he relies less on thick grafts, mucous membrane grafts, and large grafts than he once did; he now finds small thin grafts more successful. The theoretical basis for the techniques employed is the phase difference created by differential impedance of the sound wave reaching the round and oval windows. Fifty-three cases were briefly discussed as to results, and included were some reasons for early and late failures. He describes a very interesting technique in which an air-filled cavity is maintained in a newly formed middle ear cavity until healing occurs, by packing the cavity with silk thread, the end of which is brought out through the Eustachian tube and nose. This thread is pulled out through the nose after eight days, when there is less danger of adhesions between the graft and the medial wall of the middle ear cavity. Good success is claimed.

By "Paukensklerose" (the word is from Von Trollsch) is meant a special form of adhesive process in the middle ear, causing middle ear deafness. It is described histologically as showing hyaline degeneration of the connective tissue with lime salt deposits and new bone formation. It is thought to be the result of an acute, recurring otitis media in patients with an allergic constitution. Sometimes the condition is diagnosed at inspection of the drum head (lime salt deposits); very often, however, it is recognized only at operation. Especially in the attic white tissue is found, hard as cartilage, about 1-2 mm. thick, surrounding the ossicles; sometimes the attic is filled, and so the ossicles cannot move at all. These masses can be peeled off in such a way that some normal epithelium remains and movements of the ossicles are possible again. If the same masses fill out the niche of the oval window, a fenestration operation is the only possible treatment. Lime salt deposits in the drum head do not interfere with plastic closure of this membrane; moreover, they can be easily removed (the technique is described). Nine cases operated upon by Zöllner and Beck are described. Either freeing of the ossicle chain or fenestration, along with plastic closure of the drum head, was performed. The hearing improvement was very satisfactory.

An excellent work by Bozzi and Ricci deals with the physiology, the pathology, and the reconstructive operation methods for rehabilitation of hearing. Auricular dysplasia, chronic purulent otitis, tympanosclerosis, and otosclerosis are the subjects of extensive and beautifully illustrated chapters. The bibliography, filling 36 pages of two columns each, reveals the care the authors have taken in order to create a work of a high standard. This book cannot be reviewed but must be studied.

After an anatomophysiological review concerning the sound-transmitting apparatus, by Portmann and Claverio, the action of certain pathological states on the hearing function is analyzed, especially the blocking of the chain of ossicles, the immobilization of that chain, and the blocking of the round window. The effect of these various lesions are not necessarily added; on the contrary, they sometimes neutralize each other, paradoxically making hearing good. The functional operations should restore or at least maintain four indispensable anatomic conditions: a nonperforated vibratory membrane, a free tympanic cavity and a free Eustachian tube, a columella effect (contact between the vibratory membrane and the stapes), and two windows functioning properly so as to mobilize the labyrinthic fluids. Three groups of surgery permit the functional restoration: (1) stapedial mobilization for the osseous blocking, every other lesion excluded; (2) fenestration for the blocking of the tympano-ossiculary chain, whatever its cause (osseous or fibrous); (3) petromastoidectomy with or without tympanoplasties in cases where several anatomical conditions indispensable for the passage of sounds are impaired by lesions of an inflammatory kind.

The results of the classical radical operation and of the conservative radical operation are given by Pietrantoni, Bocca, and Agazzi. Improvement or deterioration of hearing is considered to have occurred when, at 500 and 2,000 cps., an increase or decrease of the keenness of hearing of at least 10 db. has been accomplished. Classical radical operation resulted in improvement (24%), deterioration (28%), and stationary (48%). Conservative radical operation resulted in improvement (29%), deterioration (21%), and stationary (50%). One must take into consideration that the stationary hearing in the cases of conservative operation does not have the same significance as in the bone centers and the preservation or even the improvement of hearing. Cases of chronic inflammation of the middle ear suitable for this are divided into three classes, according to Wullstein: (1) no affection of the ear bones, (2) affection of the ear bones affected but no affection of the stirrup, and (3) total destruction of the ear bones. Each of these three types requires a different operative technique. The results are given of partial and total tympanoplasty in 60 cases; to this the writers add another two classifications, namely, the theoretical results of each case and the social results of the operations: an improvement in 44 cases (73%), a perfect theoretical result in 14 cases (23%), and a perfect social result in 35 cases (58%).

In operations of labyrinthine fenestration for fixation of the stapes in which the osseous bridge is preserved, the functional result is restricted owing to the fact that the sound waves are halved and sent in two directions, one effectively proceeding toward the nova ovalis and the other reaching the tympanum, extinguishing itself in the ossicle chain or in the cavity, or else it is turned at the fenestra rotunda in the phase opposed to that of the movement of the sound which reaches the same fenestra rotunda via the nova ovalis. In order to circumvent this inconvenience, Bozzi has devised a technical dodge in Wullstein's operation, which makes it possible to bundle all the sound waves reaching the external ear to the nova ovalis by a modification of the shape of the operative cavity. He constructs a kind of funnel, with its narrow end on the nova ovalis which thereby acts as an ear trumpet. Finally, since the flap which constitutes the lower wall of this funnel remains loose from the tympanic

membrane and there is consequently a cushion of air between the funnel and the tympanum which forms an obstacle to the progress of the sound wave to the cavity, this results in an improvement of auditory function because the phase difference between nova ovalis and fenestra rotunda increased. Up to now the author has applied this technique to 11 operations of labyrinthine fenestration. These 11 are among the most successful of a series of 250 operations for fixation of the stapes carried out by the methods of Lempert, Aubry, and Wullstein. It is further stated that the operation is easy to perform and that the operative cavity cicatrizes rapidly and normally.

Frenckner describes a technique of plastic repair of eardrum perforations with a pedunculated skin flap of the innermost part of the auditory meatus. Satisfactory healing of the perforation and of the pathological process was obtained in 14 of 15 cases. The newly formed membrane showed a remarkable resemblance to a normal tympanic membrane. In several cases the hearing showed a considerable improvement.

According to García-Ibanez, if there exists a cholesteatomatous fistula of the horizontal canal, one should try to preserve it. If no fistula exists, the mobility of the stapes is explored. If it is mobile, a columelization by tympanostapediopexis is made. If the stapes appears to be fixed, a simple semiconservative operation is made the first time, and several months later, if the other ear is deaf, a fenestration can be made. It may be necessary to do a second-stage operation later if, after trying tympanostapediopexis, the postoperative cicatricial retraction leads to the immobilization of the stapes. The comparative audiometric examinations have shown that columelization by tympanostapediopexis gives excellent functional results with no risks to the labyrinth. In cases where it is not possible to maintain a pneumatic pad on the round window in communication with the Eustachian tube, at the expense of the tympanum, it will be possible to find a solution with the tympanoplastic reconstructive surgery of the Zöllner-Wullstein type.

The use of materials which are readily resorbed by the body is essential for good results in reconstructive surgery of the middle ear. Gelita (Gelfoam) and collagen were examined by Kley, after they were present for a time in the operative cavity. During the first week, few changes are seen. During the second week, resorption takes place. Leukocytes proceed toward the center of the foam. The author thinks that the material is dissolved by ferments and phagocytosis. Penicillin and hyaluronidase can be used to impregnate the foam. Pantothenic acid may be useful on account of its good effects on epithelization; however, it stops the action of hyaluronidase.

Kley reports excellent hearing improvement in five patients with cholesteatoma when tympanoplasty was performed. In all patients the ossicular chain was interrupted and partially destroyed. The plastic flap inserted had a small buttonhole opposite the oval window. After fenestration the author saw two patients with normal hearing instead of the expected loss of 20 to 30 db. When first starting tympanoplasties, he was inclined to build a columella of useful rests of the ossicles or to use artificial material (Supramid). Eight of twelve patients showed hearing improvement. If the artificial columella perforates the plastic flap it will most likely be discharged. Considering all their operations, he believes the hearing improvement is greater in the "window hearer" than with insertion of an artificial columella. The artificial material will set up some inflammatory reaction or adhesions. The author now never implants any kind of columella. Eroded crura of the stapes are removed. The hearing without columella, with the oval window closed by membrane or by the osseous footplate, is not worse than the postoperative hearing with complete stapes or with a new columella.

In tympanoplastic surgery, good auditory results can be obtained even with a considerably simplified sound-conduction mecha-

nism, according to Kley. In some cases the results are so good that they do not fit into the theory. Three cases of columella hearers (with either a natural or an artificial stapes) are described, in whom audiometric examination revealed only a slight limitation of tone hearing. Thus, these patients have nearly reached the theoretically calculated result, with a hearing loss of 2.5 db. Two cases of fenestrial hearers (one with preserved footplate of the stapes and one with only closure of the oval window by a membrane) are reported, in which the hearing acuity was unexpectedly good, in the latter even practically normal. These cases cannot be explained simply with the knowledge of the mechanism of sound conduction obtained so far. The second part of the publication is concerned with the efficacy of the artificial columella as recommended by Wullstein. A case is reported in which the columella of Supramid (Palavit may also be used) was expelled through the graft.

The prognosis of reconstructive operations in cases of chronic destructive otitis, is uncertain on account of the variety of adhesive processes showing themselves. Various materials used for those operations (Spongostan, Fibrospum, Sorbacel, gelatine, Gelita) which should be readily resorbed were tested by Bauer. For this test they were put into the middle ear of guinea pigs. Spongostan and Fibrospum are not totally resorbed. Gelatine is completely resorbed in 16 days, Gelita in 12 days. The author advises the use of prostheses of "resorbable" material with the utmost moderation and only the use of a material which has proved to be likely to resorb.

Fior and Sbacchi have employed in 47 cases of tympanoplastic operations a general anesthesia with a nitrous oxide-oxygen-trylene combination administered by a face piece through a semiclosed circuit. This anesthetic technique fulfills all essential requirements of this intervention and has given satisfactory results in all patients treated.

A modification of the incision for the radical mastoid operation is performed by Mikhailowski. A quadrangular skin flap is dissected, with the base in the retroauricular sulcus. Another flap, pedicled at the mastoid process, is formed from the periosteum. After the operation periosteum and skin are sutured separately, and the lines of sutures do not overlap.

A supraretroauricular incision is used by Miodonski. This is a modification of the standard postauricular incision, which gives excellent exposure of the middle ear structures. Incision begins at the anterosuperior point of insertion of the auricle and curves around the retroauricular sulcus. Temporal fascia and muscle are partly divided, partly elevated upward. Then follows transversal incision of the cutaneous meatus around its anterior, posterior, and inferior wall. Insertion of a strong retractor pulls the auricle anteriorly and inferiorly, away from the operative field.

To hasten epidermization of the radical mastoid cavity, Potapov advises the use of abraded epidermis instead of a Thiersch graft. After superficial abrasion of the skin of the upper part of the thigh he collects abraded epidermis on a small tampon of gauze saturated with hot physiologic solution and puts it on the granulating surface of the cavity. When the cavity is inspected two days later, small islands of epidermis can be seen growing on the granulating surface.

Although free autoplastic skin grafts take satisfactorily, the clinical postoperative course is unsatisfactory, reports Burian. Epithelization in radical cavities takes a considerably longer time (two to three months) than at the body surface. This is explained by the finding that in operative cavities, skin grafts shed the epidermis and the basal layer at about the second week after transplantation, whereas in grafts at the body surface the upper horny layer also peels off, but the basal layer is preserved, which makes a quicker epithelization possible. The cause of the more marked detachment of

the epidermis in radical cavities, in comparison with the conditions at the body surface, might be the higher degree of maceration of the epithelium in the humid milieu of the wound cavity. Fresh, living skin homografts are hardly used by surgeons, because such grafts are soon destroyed by antigen-antibody reactions. With stored skin, capable of regeneration, which has lost its biochemical activity, conditions are different. This takes without symptoms; it fulfills its principal function as a support and as a skeleton for the epithelization commencing from the wound edges, without any practical difference from an autograft.

The behavior of a skin flap, reports Scevola, varies in accordance with the region from which it has been taken. In order to understand such differences and, if possible, to arrive at some practical conclusions, the author studied histologically skin flaps taken from the mastoid region, the ear canal, and the internal surface of the thigh. The thickness of the epidermis is greater in the thigh, while papillae are scarce and elastic fibers are numerous. The epidermis is thinnest in the ear canal, while papillae are numerous and elastic fibers are scarce. The skin of the mastoid region is between the two above-mentioned extremes, and the author thinks that this is the kind of skin most suitable for tympanoplasty.

Nicelle and Tesco, examining the audiometric, clinical, and surgical reports of patients operated on by tympanoplasty, were able to find three kinds of audiograms which in the greatest number of cases corresponded to a particular condition in the intratympanic system: 1. Patients presenting an auditory deficit by air of 30 db. (average) showed, in most cases, a continuous and preserved ossicle chain, with good mobility. 2. Patients presented from an audiometric point of view a loss by air not exceeding 55 db. with normal or slightly impaired osseous conduction. The surgical report constantly witnessed a lesion of the ossicle chain. In this group of patients the surgical reports were varied according to the presence or absence of the ossicle chain and to its functional capacity. Equally varied is the auricular preoperative report from a clinical view. 3. Patients showed from an audiometric point of view a loss by air not exceeding 55 db. (average) with impaired bone conduction. From a surgical point of view an almost complete absence of the transmitting intratympanic system was noted, together with a preoperative auricular report that presupposed a serious anatomofunctional lesion in the structures of the middle ear. The considerations deduced by examinations of several patients have helped to foresee, although with clinical limitations, the anatomical situation of the ossicle chain and of the labyrinthine windows.

The permeability of the round window, by the introduction of 10% cocaine chlorhydrate into the tympanic cavity of 13 patients suffering from chronic suppuration of the ear, has been studied by Pennetta and Pinto. In only two cases (vertigo and hypacusia) were there symptoms of the passage of the drug through the round window membrane. The results show that in chronic suppuration of the ear there usually exists a decrease in the permeability (which most probably is accompanied by a decrease in the mobility) of the round window membrane. This fact can, therefore, be regarded as a useful test for the functional surgery of the ear.

The sound probe is a sound attached to the bone conductor of the audiometer. Audiograms are made with the sound probe placed on the drum membrane, malleus, incus, stapes, and promontory, respectively, as far as these structures are present and accessible. In cases of chronic otitis media, especially those with small perforations in the drum, comparison of these audiograms, according to Rieder and Sussmann, may show whether the ossicular chain is interrupted and whether the stapes is movable. In 48 cases operative findings confirmed the sound-probe findings.

Conduction deafness with intact drum may be caused when disease of the nasopharynx and Eustachian tubes is excluded by (1) stapes fixation, (2) adhesions with

interrupted ossicular chain, (3) spasm of the ear muscles, and (4) beginning labyrinth hydrops. Rieder and Sussmann found that the sound-probe graph in patients with intact drum or small perforations lies 20 db. above the bone conduction. Patients with stapes fixation had thresholds of probe examination which were 10-30 db. below bone conduction. Adhesions with movable stapes had thresholds similar to bone conduction or 10-20 db. higher. Adhesions with fixed stapes were below bone conduction. The exact determination of a stapes fixation is often possible with the sound-probe examination, and planning the right operation is facilitated.

Conduction deafness may arise from any part of the transmitting system (drum to stapes). Audiometry cannot give further differentiation. Thullen states that there are three components to be considered: (1) pure mechanical impediment (stapes fixation in otosclerosis), (2) impairment of acoustic-mechanical transformation of sound (large defect of drum with functioning ossicular chain) misadaptation component, and (3) pressure differences between both windows of the inner ear important for optimal hearing. The exact differentiation of the causes of a conduction deafness is imperative before any operation regarding improvement of hearing is done. The sound-probe examination is able to differentiate between acoustic-mechanical and pure mechanical loss of hearing; even quantitative measuring is possible. In normal-hearing persons the threshold of the probe sound taken from the umbo lies 20-30 db. below air conduction. In perception deafness it follows the air conduction in this distance, but in conduction deafness the graph lies closer to the air conduction or even higher and approaches the threshold of normal-hearing persons in case of normal bone conduction. Since the sound probe transmits mechanical vibration direct to the ossicular chain, the acoustic mechanical transformation is unimportant. The threshold will always lie above the air conduction as long as the ossicular chain is functioning well. When the ossicular chain is stiff, the threshold of the sound probe will approach more the air-conduction graph. The third component of pressure differences cannot be measured by the probe. In case only one window is blocked, the components of misadaptation and pressure difference appear together as the "excessive" acoustic mechanical component of hearing loss. To obtain quantitative figures we have to remember that the air-conduction threshold includes the "excessive" acoustic mechanical component and the pure mechanical one. The pathological probe-sound graph has to be compared to one 20-30 db. below the pathological air-conduction graph. The distance between this relative probe graph and the pathological one indicates the hearing loss caused by the excessive acoustic mechanical component. The difference between the pathological and the normal probe threshold corresponds to the hearing loss caused by pure mechanical impediment.

Since the introduction of the antibiotics the number of intracranial complications secondary to acute otitis media has fallen off, according to Cawthrone, and so now almost all of the complications are secondary to long-standing chronic aural infection, as in such cases the infection is so often resistant to treatment by antibiotics. When generalized meningitis or brain abscess follows chronic otitis media, the intracranial disease is the more important therapeutic problem. In the presence of intracranial complications, the otologist should be prepared to perform the classical radical mastoid operation. Anything less may well result in further intracranial infection. If the labyrinth is dead, then the operation should include a complete bony labyrinthectomy, and this may be advisable even in the presence of a fistula sign with poor hearing in the affected ear and normal hearing on the opposite side. The otologist can often prevent intracranial complications of chronic suppurative otitis media by appropriate surgery to the middle ear cleft. The only exception to this rule is when the hearing in the other ear is seriously im-

paired either by infection or by former operation.

Dingley makes a plea for early, conservative surgery (modified radical) in the treatment of cholesteatoma in the epitympanum and adjoining high posterosuperior regions. This is particularly recommended if the hearing is good. This holds true even in some cases of intracranial complications. The best hearing results are obtained if the disease is limited to the attic region, but the high posterosuperior marginal cases also frequently show good results. It is believed that it is important to preserve the incus if it appears to be functioning properly.

Bozzi and Invernizzi report the results they obtained by treating surgically fistulas caused by chronic osteitis of the labyrinth. Their surgical technique was practiced on a higher number of cases than previously considered by other authors (Timm and Tobeck) who had followed the same operative principles. Their results are as follows: 1. A careful revision of the fistula and of the osteitic area never gave origin to any diffused labyrinthitis, but, on the contrary, it constituted the essential premise to recovery from a circumscribed labyrinthithis. 2. The fistula covered by skin strips, as per Thiersch, did not show any danger of maintaining the osteitic process when properly inspected and cleaned-out in every possible site. Actually, it contributed rather on the whole to the good recovery of the operative cavity. 3. The auditory function preexisting to surgery never disappeared, but in most cases it was relatively preserved and even improved in a good percentage. 4. In most cases a decrease in vertigo was enjoyed. Anyway, they did not attain the complete abolition of the vestibular labyrinthine function.

During the 10-year period 1945-1954, a total of 856 patients with chronic suppurative otitis media were admitted to the ear clinic of the Copenhagen City Hospital, reports Permin. Of the operated-on cases, 108, or about 13%, exhibited fistular labyrinthitis. Ninety-five have been followed for periods ranging from 10 years to 4 months. Before operation, 83 had exhibited a positive fistula reaction, whereas in 12 a fistula on the lateral semicircular canal was not revealed until at operation. In all 12, the caloric reaction had been positive prior to operation. The labyrinthine lesion is of almost equal frequency in males and females and in the right and the left ear. Aural symptoms had been present for more than one year in 89 cases. Vertigo had been observed before the operation in 60. Fifty-six patients were over 40 years of age. Operation revealed cholesteatoma in 58 and only osteitis in 37. Since the operation 51 patients had not had discharge, but follow-up showed that vertigo was equally common in the absence of discharge as well as in the presence of constant or periodical discharge. Spontaneous nystagmus was found in 42 patients before the operation, directed with almost equal frequency toward as well as away from the affected ear. Acoustic function was abolished in six before the operation. At follow-up, 48 patients, or 50%, had no serviceable hearing. Labyrinthine fistula was found at operation in 62. A positive fistula reaction was elicited in 83 patients prior to operation, in 80 by compression, in 55 by aspiration with the Politzer bag, in 29 by pressure on the tragus, and in six by pressure on the vessels of the neck. It was typical in 74 cases, reversed in 8, and atypical in 1. At follow-up, the fistula reaction was present in only 25. Of 12 patients showing fistula on the lateral semicircular canal but no fistula reaction prior to operation, 4 had typical fistula reaction at follow-up. Caloric reaction was normal in 59 cases at follow-up, abolished in 10, and atypical in 26.

Costa evaluates the pathogenesis of middle ear pathology. This may be on a postinfectious basis; it may be due to allergy, or it may be a sequel to sero-otitis. He indicates that both medical and surgical treatment, particularly in the old chronic conditions, have been of negligible value in restoring useful hearing. However, he has had a fair degree of success by a transtympanic instillation of either hyaluronidase or

hydrocortisone or both. He outlines his particular technique, which is very detailed. After establishing the diagnosis that this is either a pure or mixed deafness of transmission, as a sequel of an acute or chronic otitis media or as a result of fibrosis within the middle ear cavity, he shrinks the nasal mucous membrane and the Eustachian tube orifice. Then he disinfects the external auditory canal with alcohol and thiomersal (Merthiolate). He anesthetizes the tympanic membrane with Bonain solution and then places the patient in the dorsal position and inclines the head well backward. He then makes a transtympanic injection of the first 0.75 cc. of hydrocortisone; he waits about one-half hour and makes another injection, of 0.75 cc. of hyaluronidase. The site of the injection is sealed with collodion, and the external auditory canal is plugged with cotton. He inflates the Eustachian tube with either a Politzer bag or an Itard's sound. He produces vibratory massage on the mastoid bone, followed by a short ultrasonic application, of 1 to 2 minutes, to three different sites—the first to the mastoid bone, the second from the maxillary condylar region to the opposite mastoid, and the third perpendicular to the insertion of the Eustachian tube—and, finally, a massage of the ossicles by means of a telephone and an increased and decreased intensity for a period of 15 minutes. In conclusion, he states that this work is entirely experimental but that he has had a fair degree of success.

Deafness Due to Noise, Blast and Head Trauma

High-tone deafness, according to Ireland and Hodges, results from acoustic trauma, and this can be helped by (*a*) not employing anyone in a noisy position if he has nerve deafness (This entails the testing of all new personnel by audiometry on employment.), (*b*) reducing the hazards of noise by soundproofing factories with the use of sound-absorbing materials and quiet machinery, (*c*) insistence on the wearing of simple ear wardens by the workmen, (*d*) suitable rest periods for recovery from depressed hearing in extreme levels of noise in industry. In traumatic damage to hearing by noise it seems reasonable to conclude that, if the noise is discontinued, as in the case of war veterans, the high-tone loss is not of the progressive type found in senile nerve deafness.

Fifty-six patients of different ages were examined at different time intervals (up to 36 years) after a traumatic event or the onset of the vestibular damage. Herrmann found the following facts: 1. Young persons do not show an earlier compensation than older ones, as has been affirmed by other authors. 2. Vestibular damages following head injuries show a consistent residual symptomatology after many years, with a frequency remarkably higher than vestibular damages due to other causes. This fact would be in connection, according to the author's opinion, with a central tonus difference in the vestibular systems linked to the commotio cerebri, which can be observed also in head-injured patients showing no sign of peripheral vestibular impairment.

Hearing fatigue tests are carried out by Falconnet, Portmann, and Alavoine, with the following schedule: determination of the threshold of 1,000 and 3,000 frequencies before and after the audition of white noise for 3 minutes at 100 db. One hundred normal persons and sixty noisy factory workers were tested. The transitory hearing loss observed in the latter is nearly the same as that obtained in normal subjects. Normal subjects have a rapid recovery, but some of them recover their initial threshold after a very long time. Subjects who have worked in noise for many years give the same percentage of good and bad recovery. Workers who have an occupational deafness present a similar behavior. Despite these results, it is not possible to conclude that there is not a connection between transitory and permanent hearing loss caused by the noise. In fact, occupational deafness is not produced by brief intensive trauma but by repeated and long trauma. The importance of the deficit after a very long noise dura-

tion was not investigated in this work because it is very difficult to do so in clinical practice. It may be possible to distinguish different reactions between sensitive and nonsensitive subjects. Thus, the ordinary fatigue test cannot permit the selection of workers before their employment. A too great sensitivity requires frequent examination of hearing function during the first months of work.

Since noise affects physical and mental performances as well as the auditory function, emphasis is placed by Fabian on the prevention of injurious noises. When reduction of the injurious sound to below the threshold of 70-90 phon is impossible by technical means, individual protection should be resorted to. The advantages of selectone auditory protectors are found in filtration of especially injurious high frequencies while speech perception remains intact. Subjects already affected with auditory disturbances or those who show increased acoustic sensitivity when tested should be removed from noisy occupations.

Buttner presents the results of work in reference to injury of hearing in workers in a boiler plant. He emphasizes the influence of the working conditions on both the sense of hearing and the general feeling of the workers. As a result of injurious action of noise the workers complain of buzzing sounds in the head, lasting during the period of work, and of giddiness, with nausea and vomiting. Basing on the audiometric examinations, the author has stated the dependence of hypacusis on the age of the worker and the number of years in this occupation and individual sensitivity to noise in the boiler plant.

The effect of prolonged loud noise was studied in 30 technicians whose job was the repair of airplane engines. The most frequent clinical finding, reports Bugard, apart from hypacusis, was a syndrome of general fatigue; laboratory examination showed slight alterations of the pituitary-adrenal system (hypoglycemia, eosinopenia, decreased elimination of 17-ketosteroids). From the literature it appears that such changes were also encountered with fatigue due to other reasons, and it is concluded that the metabolic disturbances found are the general, unspecific effect of the occupation. As regards EEG examinations, a high percentage of flattened curves was seen; these are not in themselves pathological, but the fact that they occur more often than is normal, and especially in the persons who showed the most marked fatigue and who had been exposed for the longest times, is still suggestive. In animals, exposure to sound and to ultrasonic waves brought about a tendency to dissociation in the EEG curves and, histologically, to edema of the cerebral cortex. On the basis of these findings, it is stated that intense noise causes specific (deafness) and aspecific (neuroendocrine) lesions. Above a critical level (160 db.) the amount of energy, irrespective of the nature of the sound, kills the animal exposed; below this level, the energy produced causes lesions deriving from the combination of emotive phenomena and mechanical and thermal trauma. There exists an adaptation to prolonged intense noise, which, together with the vibration trauma, acts on the skull and the brain, giving the effect of prolonged concussion.

Broadbent discusses some of the evidence obtained in the laboratory and in industry on the effects of noise on human behavior. The point is made that precise and scientifically valid knowledge must be used in our attempts to evaluate properly the effects of noise on man. It is shown that the noise problem is an extremely complicated one, with competing interests present that cannot always be reconciled by science. Nonetheless, it is necessary to appeal to knowledge rather than emotion in our approach to these problems. There is a brief review of the evidence with regard to effects on long-term mental health, effects on communication, and temporary effects on working efficiency.

Bostic and Worsham describe a hearing-conservation program established at a naval air station to conserve the hearing of personnel and to protect the government from

false claims. Through audiometry, a comparison was made of binaural decibel loss in two different sound environments. The number of persons suffering hearing loss at various frequencies was found to vary with their occupation and proximity to sound source. Efforts were made to reduce hazard by controlling noise at the source where possible and by providing personal protective equipment.

A relationship is shown by Aschan to exist between abnormalities in the vestibular apparatus and in the EEG. This can be of value in diagnosis of injury to nerve tissue following head injury and thus serve to help separate cases of true cerebral contusion from those of a functional nature.

After mentioning the theories of the origin of the pneumatization, the question arises as to what may be the relation between the most frequent influences, e. g., inflammation and acoustic trauma, and pneumatization of the ears. While the influence of the inflammation is decreasing, the acoustic trauma on the other hand is increasing. In order to find out whether the different degrees of pneumatization have any influence on the development of the acoustic trauma, 390 ears were exposed under equal conditions to the acoustic trauma, and the loss of hearing and pneumatization were examined. Janko came to the conclusion that, besides the different degrees of noise and length of exposure, an important part is due to the pneumatization. The impaired hearing in case of unpneumatized ears is significantly greater than in cases of pneumatized ears. From these experiments he comes to the conclusion that, at the appointment of workmen, care should be taken not only with the fatigue test, the age of workmen, and impairment of the middle ear but also with pneumatization. The percentage of the unpneumatized mastoids being rather low, the choice of the candidates would not be very difficult.

Pneumatization of the temporal bone may be a safeguard against noise deafness, write Link and Handl. A total of 166 workers with more than 10 years employment in noisy factories (80-110 db.) were examined. Of 46 workers without hearing loss, 72% had good pneumatization and 28% had poor. Of 120 workers with hearing loss, 32% had good and 68% had poor pneumatization. With good pneumatization the average maximum hearing loss at 4,000 cps was 40 db., and with poor pneumatization the loss was 60 db. Pompe reports similar results in Czechoslovakia (13.5% hearing loss with good pneumatization and 25% with poor pneumatization). Experiments showed that sound conduction was three times better through water-filled mastoid cells than through air-filled cells. Sound conduction through a sclerotic mastoid was twice as good as through a well-pneumatized mastoid. This explains the better bone conduction in sclerotic mastoids and the different bone conduction before and after fenestration surgery.

Parrack has set forth details of a research program to inquire into the control of acoustic energy and its effects on man. It is planned to satisfy four requirements. These entail varied and complex measurements and research on sources of acoustic energy, on transmission media, and on man and mechanical structures as receivers of energy. The coordination of data obtained is thus likely to produce satisfactory practical solutions. In order, for example, to determine the distance that should separate a community and an aircraft in flight, the following quantitative data are required: (*a*) noise levels produced by the aircraft during various operations, (*b*) acoustic energy absorbed by the air, (*c*) variations of the energy absorption with varying weather conditions, (*d*) a noise level acceptable to the community, to be determined with due regard for the specific activity to be carried on by each member of the community, (*e*) operational conditions, that is, the total number of daily flights, the hours of the day during which flights occur, and frequency distribution of flights throughout the day.

Systematic audiometric examinations of ground-crew personnel serving at air force

bases brought a high incidence of acoustic trauma caused by the high noise levels of idling jet engines, reports Kostelijk. Preemployment audiograms of 1,000 young applicants showed hearing impairments of more than 20 db. in one of the test frequencies (1,000, 2,000, 3,000, 4,000, 5,000, and 6,000 cps) in about 10%. Preventive measures were exclusion of persons with preemployment hearing loss, audiometric control at regular intervals, use of various forms of ear defenders. Ear plugs of impregnated cotton wool, in combination with ear muffs, according to Ruedi-Furrer, were found to give best results in combating this type of noise deafness.

From the study, by Tribel, of the audiograms collected during the yearly medical examinations of the pilots of the Belgian Air Force, it results that the jet pilots have less marked ear damage caused by noise than the pilots of piston-engined planes, other factors (age and number of flying hours) being similar. As the total intensity of the noise is higher in the jet aircraft, it is necessary to reconsider the whole problem of the occupational noise trauma of the pilot.

Measurements of the cochlear potentials, as recorded from the round window, were carried out on a series of guinea pig ears by Wever and Lawrence to ascertain the effects of injury by overstimulation with various tones. The results showed general impairments of the responses after such injury. With rare exceptions, the maximum responses to any tone are much less impaired than the sensitivity, and the maxima are more seriously affected for the low tones. In general, these changes are independent of the frequency of the tone producing the injury. These observations confirm those of earlier studies on the patterns of action of tones in the cochlea and indicate that all tones when raised to extreme levels cause widespread damage to the hair cells. It is suggested that certain hair cells, which are normally involved in the peak actions of all tones, are particularly susceptible to damage by overstimulation.

Adaptation, fatigue, and acoustic trauma are three separate entities. Tanner has demonstrated in his material that a strong adaptation goes hand in hand with a small fatigue and vice versa. Adaptation thus seems to be a means of autoprotection of the ear against persistent damage. Major individual differences suggest the possibility of screening out vulnerable ears. The latter are especially found among sympathicotonic persons. The marked adaptation found in parasympathicotonici, as well as the "deficient" adaptation in sympathicotonici, shows marked influence from parasympathicolytic and sympathicolytic drugs.

Two groups of rabbits, one group consisting of normal animals and the other of rabbits in deep anesthesia, were exposed to sound trauma of the intensity of 100 phon for five hours per day for periods varying from 15 to 30 days. The purpose of the experiment, reports Takahashi, was to determine whether the suppression of the response tonus and of the reflex contractions of the muscles of the middle ear, caused by the deep urethan anesthesia, would lead to cochlear lesions in the ears of the anesthesized animals different from those obtained in normal rabbits under the same experimental conditions. It was possible to demonstrate, by seriated histological preparation, that the atrophic cochlear degenerative lesions were much severer in animals given an anesthetic than in animals exposed to the sounds of waking conditions. From these experiments it is concluded that the muscles of the middle ear exert a protective effect on the inner ear against the damage caused by repeated exposure to sounds of 100 phon intensity.

An experimental study was carried out, by Takahashi, on 16 healthy rabbits subjected in the first stage to intracranial resection of the trigeminal nerve on the right and then exposed to explosive acoustic trauma of both ears. The purpose of this experiment was to determine whether there were differences between the histopathological damage to the cochlea caused by the acoustic trauma in the ear with the intact

trigeminal nerve as compared with the ear where the trigeminal innervation was lacking. The histopathological study, illustrated with six photomicrographs, reveals that the degenerative cochlear lesions are much severer on the side where, because of the lack of trigeminal innervation, the protective function of the tensor tympani muscle was less. The great severity of the destructive lesions of the organ of Corti is attributed to the lack of normal response tonus and of reflex contractions of the tensor tympani muscle, caused by the division of the trigeminal nerve. This study confirms that the tympanic muscle serves to protect the cochlear structures from too intense sound stimulation.

Every acoustic stress of the organ of Corti is followed by a loss of hearing with recruitment, reports Lerche. The reversible loss is called "fatigue," the irreversible loss is called "sound trauma." Continued registration of the microphon potential taken from the round window of cats revealed the following facts: 1. The bioelectric sensitivity of the cells of Corti's organ decreases under the influence of acoustic stress. 2. The decrease of sensitivity is less or not marked regarding loud sounds but is well marked regarding weaker sound stimulation. 3. This bioelectric attitude of the cells corresponds with the attitude of the ear regarding fatigue or adaptation. Here also the loss of hearing is more marked toward weak sound stimulations than toward the louder ones (recruitment).

Five untrained listeners were tested by Hirsh and Bilger with a modified Bekesy audiometer in order to observe threshold recovery following exposure to a 1,000 cps tone at sensation levels of 20, 60, 80, 90, and 100 db. and at durations ranging from 10 seconds to 4 minutes. Threshold recoveries were noted for both 1,000 cps and 1,400 cps to allow for a detailed study of the relation between hearing loss following exposure to pure tones and the level and duration of the exposure tones. Families of curves are presented, showing recovery functions for the parameters studied, while analyses of variance show both sensation level and duration, as well as time of measurements, to be significant sources of variation. Among other conclusions, the authors note that the loss for 1,400 cps is more clearly related to duration and level of exposure than is the loss for the 1,000 cps test tone. In addition, it is noted that a greater portion of the total loss at 1,000 cps is recovered during an early phase of recovery than is the case at 1,400 cps. A lower threshold value than that preceding exposure was found for some durations and levels of exposure at 1,000 cps only. The authors suggest that the particular patterns of recovery shown by the curves suggest more than one underlying recovery process.

In an excellent controlled study of per-stimulatory auditory fatigue, Carterette has presented methods and has added information concerning the adaptation of the ear to the stimulations used. It is shown that interrupted fatiguing noise and continuous fatiguing noise have much the same effect, with the greatest fatigue occurring during the first minute of stimulation.

Bernabei reports his functional and histological research of an acoustic experimental trauma, produced by the white noise of "Larderello" boracic geysers. The author establishes and explains (1) the systemic localization of the greatest injuries in the section between the end of the first and the beginning of the second cochlear coil, (2) the correlation between the experimental histological injury and the functional acoustic alteration of the workers injured by the same trauma, (3) no histological cochlear injuries when the intensity of noise is less than 96 db., and (4) the progress of the degenerative injury from the auditory cells of Corti's organ to the central cochlear nerve on long exposure to the noise.

Examinations of the statoacoustic apparatus in 29 amateur boxers, by Krajina and Milojevic, proved that 50% of the cases had hearing disturbances. These disturbances were of a slight to moderately severe degree. After comparison of this

lesion with the EEG findings it was decided that these injuries are mainly of a peripheral nature. They are caused in particular by mandibular K. O., in which the trauma spreads directly through the ossicles to the temporal bone or to the labyrinth. The lesions are mostly localized in the cochlea; disturbances in the vestibular portion are less frequent and were minimal in the present material.

Having examined 26 cases of rupture of the eardrum due to explosions, Paldi described the well-known symptomatology of these accidents. Significantly new is his observation that a traumatic perforation of the drum does not necessarily protect the inner ear from the acoustic trauma. The recovery of hearing depends on the audiometric hearing loss found after the accident and on the clinical course. These findings underline again the necessity of providing with ear protectors all persons exposed to loud noises or explosions.

Bourgeois discusses cranial trauma and its relation to the temporal bone. Fractures of the skull present few symptoms in the external meatus. However, pathology due to trauma of the skull can be evidenced by lesions of the tympanic membrane and hemorrhage in the tympanic cavity. These may eventually influence hearing, and it is extremely important to evaluate the presence or absence of a rupture of the tympanic membrane, particularly from the medicolegal standpoint. Secondary infections may also occur. The prognosis is important, and it is influenced by (*a*) disruption of the ossicular chain, (*b*) cicatricial process, (*c*) adhesions in the middle ear, and (*d*) presence of secondary infections. Longitudinal fractures of the temporal bone produce auditory impairment, particularly of the upper tones. Perpendicular fractures usually produce total deafness, with an unresponsive labyrinth to the caloric test. The author also discusses deafness in cranial injuries without fracture. This deafness is usually bilateral, and it usually involves the upper registers. Equilibrium is also affected by cranial injuries. It may be either of a peripheral nature, wherein the symptoms are temporary and easily compensated, or of a central nature, wherein the symptoms are prolonged and compensation is poor. In perpendicular fractures of the temporal bone or fractures in a paralabyrinthine area there is unilateral destruction of the labyrinth, which is total and is associated with total deafness. The author also discusses the postconcussional symptoms, which may be due to a temporary edema of the cerebromeninges: 1. The lesions may be benign, where there is a bilateral hyperexcitability of the labyrinth. 2. In other lesions there is a cerebral contusion, and the symptoms are more serious and are accompanied by neurologic manifestations, such as hemiplegia, monoplegia, and involvement of cranial nerves. Here the vestibular signs can be extremely variable. 3. Radicular lesions are usually unilateral, with the hearing and a perception type of deafness without recruitment and a hypoexcitability of the vestibule.

Italian legislation grants a pension of up to 90% of the working capacity of the subject in case of hearing loss and up to 100% when it is followed by a damage to speech, reports Frugoni. Only the U. S. military administration provides a similar treatment. The only valuation of the deficit quantum recognized by law is given by the classical vocal audiometry at a variable distance. This technique has two great advantages—the human voice represents the most physiological stimulus to the ear, and no special equipment is required for the test. On the other hand, many are the inconveniences of this technique. In case no equipment for the vocal audiometry is available, the Fowler method is universally recognized as an excellent one for supplying a satisfactory judgment. Unfortunately the Italian language is very rich in vowels, and so low and middle frequencies are more frequent than in the English language, in which Fowler carried out his tests. When a normal audiogram by air and osseous way is drawn, the hearing deficit is valued for each ear according to the audiometric data obtained. These data can be turned

into metric distance according to the table that Frugoni drew after examining 250 cases, as the Watson and Maguire tables proved to be unsatisfactory for the Italian language. It was objected that the results obtained were not exact, but the tables on which war pensions were based by law allowed rather wide limits. The tests previously used were much less exact, and so it often happened that the disabled who were examined were entered at different times and in different categories even by the same medical commission.

Toxic Labyrinthitis

Streptomycin cannot be used without proper indication because of its toxic influence on the eighth nerve, warns Escribano, Garcia, Sola. Corti's organ, utricle, and saccula maculae and cristae are the affected zones. The drug acts in the same way as neurotropic viruses, spreading in the endolymph, where it produces severe troubles in the metabolism of auditory cells, starting in the first spiral of the cochlea with loss of its high tones. This action over the nerve endings has been made use of in some cases when hyperactivity had to be reduced. In spite of several proposed treatments, streptomycin deafness remains hopeless.

The sole toxic effect in therapeutic applications of streptomycin is the damage caused in the vestibular apparatus, while dihydrostreptomycin injures the acoustic apparatus. Entity and frequency of the lesions are strictly dependent on the hepatic and renal functions. After examinations and experiences with different groups of guinea pigs, it was concluded by Fachinelli that isonicotinic hydrazide cannot modify the vestibular toxicity of streptomycin. The methanesulfonic derivate, on the other hand, seems to provoke an experimentally ascertained reduction of the damage. A purely diuretic action of the sodiomethanesulfonic of isoniazid is to be excluded, and so it is still to be cleared up which action causes a reduction in the toxicity.

Late streptomycin damage of the cochlear nerve is reported by Vyslonzil. A preexisting nerve deafness may be aggravated by dihydrostreptomycin. Renal insufficiency predisposes to cochlear damage by use of streptomycin. Deafness was seen in meningitis and intrathecal therapy with dihydrostreptomycin but not with streptomycin. Deafness did not occur with intramuscular injections in miliary or cavernous pulmonary tuberculosis.

Toxic effects of streptomycin and dihydrostreptomycin are reviewed by Vyslonzil. Waltner saw 93 patients treated for three months with different doses of streptomycin. With 3 gm. of streptomycin there was damage of the vestibularis in 74 and damage of the cochlearis in 15; with 1 gm., the corresponding numbers were 32 and 9, and with 0.5 gm., 3 and 0. In prolonged treatment no damage was noted with doses of 16 mg. of streptomycin per kilogram of body weight in adults. Children under 10 years of age tolerate 24 mg. per kilogram without cochlear damage. Dihydrostreptomycin affects chiefly the cochlear nerve. Vestibular damage may occur a few days before or at the same time. Streptomycin affects first the vestibular nerve. Dihydrostreptomycin should be given only I. M. and not more than 1 gm. daily and not too long. Retarded excretion in renal disorders has to be kept in mind. Experiments have shown the first damage to be flattening of the labyrinthine nerve cells (hair cells). An unspecific meningitis may occur, which affects the eighth cranial nerve alone. Most of these patients have toxic conditions, and the sum of this damage and the alteration of the hair cells by streptomycin may lead to a sudden breakdown of their function. Pediatricians found that hearing loss occurred first in the ear on which the children sleep. Changes in the blood supply may play a certain role. In animal experiments, Baroni, Zancucchi, and Masera found the streptomycin level in the endolymph to be twice as high as in the blood. Therapy should try to facilitate the outflow of the labyrinth fluid. This may be done by lumbar punctures or blockage of the ganglia.

The ingestion of 200 acetylsalicylic acid tablets in six days was followed one day later by a severe bilateral perceptive type hearing loss and evidence of recruitment, plus recurrent tinnitus and recurrent sensation of unsteadiness. The hearing returned to normal one week later. The findings are explained by Waltner by increased intralabyrinthine fluids and pressure with immobilization of both windows and with inactivation of the external hair cells.

Taniewski refers to his earlier paper (*Med. pracy* 1:11, 1952) on toxic effects of CS_2 on human beings. In this paper he reported on the results of examination of people working in the artificial silk factory, in which CS_2 is used. From the results of the vestibular tests he came to the conclusion that the disturbances of equilibrium, often encountered in the patients with chronic CS_2 intoxication, are caused mainly by changes in the C. N. S. In the present paper the results of experiments upon animals are given. In an artificial silk factory there are always slight traces of CS_2 in the atmosphere although the content of CS_2 in the air is constantly kept under control. A cage with six guinea pigs was placed in such a factory. After 8-33 days all animals died, and their brains and ears were examined histopathologically. No significant changes could be found in the inner ears, whereas in the brain of all guinea pigs such changes as extensive hyaline thromboses of small vessels, inflammatory infiltrations around the vessels, and small necrotic foci were seen.

Acute alcoholism has no influence on hearing. A vestibular symptom was positional nystagmus with varying direction, therefore of central origin. Together with the elimination of the alcohol the direction of the positional nystagmus is reversed. The latent period of the caloric reaction is often shortened considerably. Schwab and Ey discuss the possibility of using these data for the evaluation of drunkenness.

Administration of chlorpromazine caused no variations either in subjects with normal auditory functions or in patients with chronic otitis media, report Pinto and Pennetta. In patients with hypacusia due to labyrinthine lesions, however, it caused constantly a decrease of the auditory capacity of from 10 to 40 db. for high frequencies. This effect can be explained by taking into account the so-called narcobiotic effect of the drug, which is thought to be more profound in subjects with pathological conditions of the cochlea.

Based on clinical observation in a case of fluoroacetate poisoning, animal experiments were carried out by Kelemen, Ambrus, and Ambrus to observe the effects, if any, of this antimitotic metabolic poison on the hearing organ. Against a background of residual, quiescent changes, as encountered in the middle ear of most laboratory animals, the following pathological pattern develops under the effect of acute fluoroacetate poisoning: engorgement of all vessels in the middle ear and internal ear and transformation of the organized old residua in the middle ear into the product of an acute recrudescence in the form of hemorrhagic suppurative otitis media. This is followed in many cases by induced, collateral, serofibrinous otitis interna, with scattered, petechial bleedings in the inner ear. Given more time, the development of suppurative panotitis would be highly probable. Since nothing was known about the reaction of the tissues of the hearing organ to fluoroacetate, in this study high, quickly lethal doses were used, to render any changes more conspicuous.

The ear in chronic benzene poisoning was reported by Costa and Mazzei. In 20% of the cases an auditory deficiency of perceptive type occurred, especially at the level of the frequency 4,096. The experimental studies of the auditory organs in rabbits subjected to chronic poisoning with benzene vapors revealed the development of cochlear alterations of degenerative type, involving particularly the neurosensorial elements, the ganglia of Scarpa and of Corti, and the nerve trunks, usually secondary to lesions

of the vascular system. Hemorrhagic and serous effusions were demonstrated in the cochlear canal, in the tympanic and vestibular spaces, and in other anatomical formations (vascular stria, spiral ligament, organ of Corti). Histological investigation of the bulb showed a marked vascular congestion, with hemorrhagic stippling, especially in the vicinity of the meningeal lamina, and degenerative phenomena of the cells of the bulbar acoustic nuclei. It proves the possibility that chronic benzene poisoning affects also the auditory organ, producing an acoustic deficiency of purely perceptive type, a fact that is of importance in human occupational pathology.

Audiometric and vestibular examinations were undertaken by Ciurlo and Ottoboni to determine the functional condition of the inner ear in 50 laborers who had been exposed to lead for long periods of time. None of the subjects examined showed vestibular disturbances of any importance, but auditory deficiency was found, especially with regard to high tones; there was no correlation between the auditory deficiency and the severity of the toxic manifestations of general nature. The diminution of hearing develops slowly and progressively.

A rather high percentage of patients treated with hydrazide develop vertigo and tinnitis. Ancetti reports a progressive lowering of the postrotatory nystagmus following angular acceleration (of a constant physical value) in 80% of the cases; it was impossible to ascertain both the residence of the toxic lesion (peripheral or central) and the relation existing between its entity and the functional modifications noted. The experimental material used by Pivotti in his study of the vestibular activity following the administration of the isonicotinic acid hydrazide was submitted by the author to a histological test either on the cocheovestibular peripheral organs or to the cochlear or vestibular nucleus. Isonicotinic acid hydrazide (piridin 4-carbosil hydrozide) administered to guinea pigs in toxic doses and during a long time causes some alterations in the vestibular activity, which corresponds to histopathological lesions mainly on the cochlear and vestibular nucleus, varying in entity according to the administered doses. These lesions are less dangerous than lesions caused by other drugs toxic to the vestibular system, such as streptomycin and quinine or salicylate; these data might have some importance in the extended therapeutic use of isonicotinic hydrazide.

Combined treatment of optobine with dihydrostreptomycin was applied in five cases of tuberculosis otitis media in daily doses of ½-10 grains (0.032-0.65 gm.) with good results. Castello and Cremonesi contend that optobine has a favorable catalytic enzyme-like influence on human tissues and exerts a protective action on the labyrinth against streptomycin damage.

Roberto and Bellomo describe a patient who was supposed to undergo a surgical intervention for otomastoiditis while actually the symptomatology was due to a middle ear tuberculosis. Recovery was obtained after two months with a treatment of streptomycin, PAS, and isoniazide, and a considerable hearing gain ensued after a year. The authors venture a hypothesis on the mechanism through which streptomycin was able to improve the hearing function of the patient. It is surprising to notice how a hearing gain was obtained by means of a drug, such as streptomycin and in particular in its dihydric form, well known for its toxicity to the ways and nucleus of the acoustic organ. This result might be explained, considering the action that streptomycin might have on the Koch bacilli, by a cytochemical mechanism similar to the one remarked in the cellular elements of the ear following its use: the tuberculosis bacillus (or its toxins), localized at the ear's level, works on the nucleoprotein exchange of the nervous cells, thus interfering in their synthetic capacity either by inhibiting action of the enzyme systems there deputed or by anomalous consumption of the nuclear acids already synthetized. There would result a reduction in the cytoplasmic contents of nuclear acids with a proportional loss of

hearing capacity. The streptomycin would cause normalization in the nucleoprotein exchange by the liberation of the nuclear cytoplasmic acids following neutralization of the tubercular toxins (which would bind to the streptomycin) with a contemporary and proportional restoring of the hearing function.

Inflammatory Diseases of Labyrinth

After some remarks on the problem of morphobiological identity between the virus of chickenpox and the one of herpes zoster, Piatti concludes that this problem is still to be solved. Then he underlines the general symptoms, the cutaneous and mucous signs of herpes zoster, which is an infectious ganglioneuritis whose pathological lesions may be found at the level of the affected skin as well as in various segments of the sensitive neuron. Hunt's old classification of herpes zoster of the head is maintained by the author: herpes facialis, oticus, pharyngis, laryngis, and occipitocollaris are described and their symptoms illustrated. The therapy may be either local, cutaneous and mucous, or general. When the skin blisters are intact and separated, an inactive powder should be applied (50% venitian talc and 50% zinc oxide) with a possible addition of 5%-10% anestesine. When blisters are very big and confluent, it is advisable to empty them aseptically before powdering. Compresses and ointments (though not advisable) should be adopted only in case of intense edema or ulcerative and gangrenous process. As a general therapy different treatments were adopted, ranging from common antirheumatic drugs to vitamins or antibiotics drugs. Thiamine (vitamin B_1) in big doses (100 mg. pro die) is much used today, together with its pyrophosphoric ester or cocarboxylase, administered either by intramuscular injections (25, 50, 100 mg. pro die) or intravenously (20 mg. pro die for 6-10 days), giving rather satisfactory results (Tinozzi and others). Also cyanocobalamin (vitamin B_{12}), which is probably present in the biochemistry of the nervous cell, proved to be useful in doses ranging from 30μg. to 500μg. and 1,000μg. pro die intramuscularly (Grasso, Colucci). Zoster was also treated by the intramuscular administration of (Nickel) or by the administration of dicorticotropin (10-15 mg. every six hours) hydroergotamine (1 cc. intramuscularly or intravenously for four to six days) (Combes, Canizares, Simuango). In particularly debilitated cases it might be useful to administer hepatic extract parenterally, together with cyanocobalamin. Antibiotics have been used against herpes zoster, and the most active one proved to be aureomycin. Should pains become unbearable it is necessary to have recourse to common analgesic or antineuralgic drugs. In some, alcoholization of the ganglion or nerve proves beneficial or even the surgical interruption of the sensitive neurone.

A detailed description of a case of Hunt's syndrome is given by Rochat and Terrier. Recruitment, even overrecruitment, was found. This seems to support the assumption of an intracochlear herpetic eruption as a homologue of the skin manifestations and cause of the deafness. When these latter are absent a differential diagnosis with "idiopathic" Ménière's will sometimes prove difficult.

A case of herpes zoster of the ear is discussed by Glaninger. The audiometric finding, which first showed a positive and later a negative recruitment, led the author to the opinion that the affection was primarily localized in the labyrinth and later spread upward.

Nager reports an examination of the temporal bones of two persons, who were very deaf in life, who were observed for a long time and treated for lues hereditaria. Macroscopically, there were no signs of the disease. Histological examination showed a chronic nonsuppurative osteomyelitis of the petrous and tympanic portions with invasion of the labyrinth and other typical changes. The subacute invasion of the labyrinth with subsequent neurolabyrinthitis explains the intermittent progress of this disease. The

possibility of a hematogenetic origin of the osteomyelitis is suggested.

Ladyzhenskaia reports two cases of long-standing tertiary malaria in which stormy vestibular symptoms were observed during typical attacks of malaria. Between the attacks the general condition of both patients was very good. No parasites were found in the blood. The first patient had only slight hearing loss and tinnitus in her left ear; the vestibular reactions to both caloric and rotation tests were normal. Before the attack there appeared chills and generalized urticaria of the skin; then the temperature rose to 40 C (104 F) or more. During the attack the patient had marked rotatory dizziness and nausea; at the same time spontaneous horizontal nystagmus to the left, of medium amplitude, was noted. In the course of a long clinical observation, dizziness during the attacks became rather nonrotatory and the patient often vomited. The direction of the nystagmus was then very variable—horizontal to the right, rotatory, or vertical. Tinnitus became more intensive, hearing loss in the left ear greater. Treatment (Chinin, thiamine, Urotropin) brought no improvement. In the second case, hearing loss in the left ear was greater and the left labyrinth was hypoactive. During the attack, a coarse, tonic nystagmus with a vertical component was noted. In the author's experience of 600 treated cases of malaria, some changes in the vestibular apparatus, during the attacks and between them, are often encountered. In his opinion, these changes are the result of repeated, abnormal stimulation of the C. N. S. by each attack of malaria.

A premature infant (7 months) was born on the 40th day of the mother's poliomyelitis and died 44 hours after birth. No microscopic evidence of poliomyelitis could be found in the infant's neuromuscular system by Kelemen. A subarachnoid hemorrhage, a pulmonary hemorrhage, and a massive hemorrhage of the middle and inner ear were found. This suggests vascular damage due to perinatal causes. The obstetric trauma to the ear is ascribed to the prematurity and perhaps also to anoxia as a consequence of the poliomyelitis.

Cochlear troubles in brucellosis are rare, according to Faidherbe, and can be found in the chronic phase of the disease. In most cases they are complicated by meningeal involvement but can be isolated also. Hypacusia of reception and labyrinthic hyperexcitability are the first signs of the eighth nerve involvement and are probably due to angiospasms. Antibiotics, especially chloramphenicol and chlortetracycline, are of the best therapeutical value. It is recommended that vasodilatators of cerebral vessels be tried.

In one-third of the cases of brucellosis, a lesion of the auditory nerve can be established which is situated either in its peripheral or in its central portion, i. e., in Corti's organ, in the auditory nerve, in the nuclei, or in the centers. The present studies, by Remorino, Thompson, and Molinelli, were made in a large number of brucellosis patients, in whom this disease was verified by appropriate laboratory examinations. Audiometrical, clinical, otoneurological, and laboratory examinations (C. S. F.) were carried out. In only two cases were pathological changes established in the C. S. F.; reference is made to the scant literature on affections of the ear due to brucellosis and its pathology. In the vestibular apparatus, peripheral (labyrinthine) and central disturbances were demonstrated, and occasional cases of isolated lesions of the vestibular nerve, with completely normal function of the cochlear nerve, were presented.

Eigler states that cochlear damage in virus infections, such as measles and mumps, may be due to meningitis, neuritis of the acoustic nerve, or labyrinthitis. In examining 21 patients recovered from severe virus meningitis, he found no hearing losses. In 36 patients recovered from bacterial meningitis, 2 were found with total deafness (meningococcal) and 1 with hair-cell damage (streptococcal).

Some facts, according to Clement, emerging from observations of several epidemics

of rubella, bearing on the importance of the time element in rubellar embryopathy are discussed; e. g., when infection occurs during the sixth week of pregnancy, the eye, heart, and several other organs may be involved. As a prophylactic measure simple exposure is too uncertain, inoculation of rhinopharyngeal secretions or of serum from a patient during the first days of the eruption is recommended and is followed in a high percentage of cases by the development of the disease after a mean incubation period of 12 days. Passive immunization gives short protection. The conflicting results obtained with γ-globulin are discussed. The problem of abortion is considered.

Acoustic and vestibular tests were carried out, by Hallet and Janssen, in 11 patients affected with human trypanosomiasis (sleeping sickness), and caloric reflexes were found to have disappeared in many cases both to cold (50% of the cases) and to warm stimulation (19 ears out of 22). Galvanic tests, however, always revealed a certain hyperexcitability. The results of these tests are suggestive of lesions of the central vestibular pathways.

Transitory unilateral disorders of tone perception in influenzal otitis media are reviewed by Hohlbrugger. Tone perception is shifted to one-half octave higher due either to stiffening of the basilar membrane or to increased viscosity of perilymph. This disturbance is seldom detected, because most people do not differentiate half tones.

Deafness in Children

The audiograms for 250 cases of congenital deafness were related as to slope and general type to the causes of deafness within the congenital group. Three main types of audiograms were found, labeled the flat, sloping, and residual group. There were also suggested three main types of causes. In one, pathological agents acted upon the undeveloped apparatus during embryonal life. In the second, the agent acted on a fully developed organ of hearing. In the third group, the development had been arrested, presumably by genetic factors. X^2 Tests of the associations among these groups were significant. The results of the analyses led Fisch to suggest that developmental arrest in the organ of Corti as a whole leads to a flat type of loss while deafness due to pathological conditions at birth or in the immediate postnatal period lead to selective loss for the high frequencies. The association of the residual type of graph with hereditary deafness was also found to be significant. However, other types of audiograms found in the hereditary group suggested that it would be interesting to examine the possibility of a fundamentally different pathology in the various types of deafness with family history, which so far are classified as one large hereditary group.

Barr reports the results of an investigation comparing pure-tone audiometry by means of the PGSR (psychogalvanic skin resistance) and "play audiometry" in a series of 324 children under the age of 7 years. The first two chapters review the various methods that have been used. This is followed by descriptions of equipment and technique during both PGSR and play audiometry. The 324 children tested were divided into three groups: (1) those who responded to sound, (2) those who did not respond to sound, and (3) those with grave general retardation and/or behavior problems. In the first and second groups, there was close correlation between the results obtained with both pure-tone and play audiometry, and the information reported indicates the advantages of the two methods. Play audiometry could be done in children 2½ years of age or older, while the PGSR method could be employed with children less than 2 years of age. The conclusion drawn by the author is that, since play audiometry is pleasant for the patient while PGSR audiometry is unpleasant, play audiometry should always be attempted first, assuming that the child is old enough that successful results can be anticipated. Because of the complexity of the PGSR

method it might expediently be reserved solely for the cases in which play audiometry has failed.

Of 965 children seen at the Center for Children with Hearing and Speech Defects, in Warsaw, 766 deaf and hard of hearing were thoroughly examined to establish the cause of deafness, reports Siedlanowska. In addition, 160 children from the lower classes of the Institute for the Deaf were also examined. Altogether, the case material consisted of 925 children, of which the great majority was 6-8 years of age. It was divided into three groups: (1) inherited deafness, (2) developmental anomalies, and (3) acquired deafness. The last group was subdivided into three subgroups, depending on the time when the harmful factor took effect: (*a*) during intrauterine life, (*b*) at birth, or (*c*) after birth. Inherited deafness was found in 126 cases; developmental anomaly, in 14 cases; acquired deafness, in 498 cases; in 288 cases (31%) the cause of deafness could not be established. The causes of acquired deafness were listed as follows: meningitis (271 cases), head trauma (38 cases), healed otitis media (34 cases), active otitis media (34 cases), scarlet fever (32 cases), and other infectious diseases.

Manzini, Marenzi, and Teseo underline the importance of collective audiometry for the selective research of functional hearing losses on school children and refer the results obtained in 59,850 Milan primary school children. The total number of hypoacousias observed was 3,075 of 59,850 children tested, with a frequency of 5.1%. These hearing losses have been divided according to their degree into 2,415 slight, 540 middle, and 120 severe cases. The authors consider finally the prophylactic and therapeutic possibilities for each group of hearing losses observed and conclude that collective audiometry in school children has a great clinical, social, and pedagogical importance.

The audiometrical examinations differ according to the mental age and not the real age of the child, according to Grimoud. In children older than 7 years, audiometry is carried out as in adults. In children between 3 and 7 years of age, tonal or vocal audiometry is applied, sometimes with the aid of Dix and Hallpike's "peep show" method. In children under the age of 3 years, vocal audiometry with the "Response to the Christian name" is used. The best results are obtained with objective tone audiometry or psychogalvanometry.

In a genetic analysis of 141 deaf children, no cause could be determined by Hata in the largest single group; children with a hereditary background were the next group. The Rh factor was not frequent as an etiological factor in Hawaii. The importance of the genetic history is emphasized.

An examination by Cheeseman and Stevenson of the Irish census enumerations from 1851 to 1911, with particular reference to the age distributions available for 1881, 1901, and 1911, gives no indication of any generation of people in whom a particularly high proportion were born deaf. Similarly, admission records of a school for the deaf fail to reveal evidence of an unduly large number of children having been born deaf in any particular year or calendar month. These findings are of interest, as presumptive evidence exists that in about one-fourth of the deaf-born persons at present living in Northern Ireland the deafness was not of genetic origin. The hypothesis that some of this nongenetically determined deafness is attributable to maternal infections in early pregnancy is thus not supported by the census and school data. However, they cannot exclude such a hypothesis because of their inherent limitations. Because the deafness of genetic and nongenetic origin cannot be efficiently distinguished, direct examination of the latter is not possible in the majority of cases. However, the distribution by rank and season of birth of a group of living deaf-born persons with unaffected sibs and parents, of whom probably one-half are deaf from nongenetic causes, could easily have occurred by chance.

Team work in the therapy of alexia is strongly emphasized by Van Luijpen. The collaboration of speech therapist, neurologist, psychiatrist, pediatrician, psychologist, social worker, otologist, and audiologist is necessary. Sixty-five children with alexia were tested with Van Dishoeck's continuaudiometer. Twenty per cent showed a dip of 10-30 db. in different frequencies. Functional differences between the left and right ear were found in about 60%. The author found in most cases a so-called acoustical lability. In other cases an auditory imperception was found. The dynamograms showed very sensitive ears in the cases of acoustical lability and unresponsive ears in those of auditory imperception.

An audiometric survey of 89 cerebral-palsied children from three schools showed that 20% of them had a significant degree of deafness. A further 14% had a significant hearing loss in at least two adjacent frequencies. The difficulties encountered in carrying out such tests are discussed by Fisch. It is felt that all cerebral-palsied children should have a detailed hearing test.

Riecker and others give a report of clinical and audiometric examination of 79 pupils in a school for the deaf. The possibility of an electric acoustic speech amplifier was especially checked; with amplification 19 pupils showed a useful speech hearing and speech understanding at once; 18 pupils had no speech understanding. They might get some acoustic perception by training. In 37 pupils hearing aids were estimated to be of no use.

Walter attempts to show the order, if any, in which sentence constructions and parts of speech appear to be mastered by profoundly deaf children. Such an order being found to exist, an attempt is made to find the factors associated with the deaf children's mastery of these language forms. One hundred two children were tested. This was the total number of children aged 6 years to 12 years 11 months who had had a hearing loss of 80 db. or more since birth and who were in attendance at a school for deaf children in New South Wales. The children were required to write sentences about simple pictures. Five stages were apparent in the unaided sentence construction of this group of profoundly deaf children. 1. The most advanced stage could be determined by ability to write a correct compound sentence. 2 The second highest stage could be determined by ability to write a correct simple sentence and by attempts to write compound sentences. 3. The middle stage appeared to be a transitional stage in which children either confined themselves to simple sentences or attempted to use compound sentences. 4. The second lowest stage could be recognized by attempts to use simple sentences only. 5. Nouns and articles only were used; written sentence construction could not be said to have begun in this lowest stage. The order of emergence of various constructions and parts of speech in the written language of these profoundly deaf children appeared to be: (1) nouns and articles, (2) verbs and prepositions, (3) conjunctions and pronouns, and (4) apostrophe and direct speech. It seemed that, in a school beginner who had been profoundly deaf since birth, his I. Q., as measured by the Nebraska Test of Learning Aptitude, and his chronological age at the time of enrollment were the factors likely to give some indication of his potential ability to learn language.

A school health program comprised the otological and audiological examination by Bordley and Hardy of approximately 5,000 children. Of these children, 582 (10.7%) showed initial audiological abnormalities. These children were examined for 5 years. All of these children were instructed as to the prevention of infections of the upper respiratory tract in swimming, etc. Half of them were treated with radium and the others with a placebo. The lateral areas of the adenoid and the peritubal edema were especially diminished during the first 18 months after the irradiation. Regression of the adenoid was quicker in irradiated children than in nonirradiated ones. During the observation period, the peritubal adenoid showed little reduction in the control chil-

dren, and so the radium irradiation is concluded to have been successful. The irradiated as well as the nonirradiated group showed improved hearing, but the irradiated group to a higher degree. In the nonirradiated group this improvement must be attributed to increase of age, habituation to the examination, and improved nasopharyngeal hygiene. This experiment thus proves that irradiation of the tubal orifice is an important factor in the prevention of audiological abnormalities in children.

Thirty-six hard of hearing students in the seventh, eighth, and ninth grades, demonstrating a minimum hearing loss of 15 db. or better at each of 512, 1,024, and 2,048 cycles or 20 db. loss at any two of these frequencies or 25 db. loss at any frequency were studied for their adjustment and school environment by use of the California Achievement Test and the California Test of Personality, and by rating by various teachers. These 36 hard of hearing children were studied by the matched-pair method with normal children. In this study, the children with minimal hearing loss adjusted to their school environment as favorably as their normal-hearing partners, according to Reynolds.

Wolferman is chiefly concerned with the differential diagnosis between congenital auditory aphasia and deafness. Though he touches upon other causes of congenital aphasia, such as amentia, emotional disturbance, and peripheral paralysis, most of the article is taken up with the problem of evaluating the hearing of the child who is late in the acquisition of speech comprehension and production. The author discusses in particular detail the testing of hearing by means of the psychogalvanic reflex. Though he considers, "This test appears to be a powerful weapon in the diagnosis of these cases and should be employed freely in young children with hearing and speech difficulties," he calls attention to the fact, "A normal psychogalvanic audiogram suggests integrity of the auditory pathways up to the brain stem, but is of little help in the localization of lesions above this level." Thus he maintains that some children who, with this test, seem to hear actually do not, if we define hearing as an awareness of sound. The author considers that there are many more congenital aphasics than the few who are recognized. He regrets that these unrecognized trainable children are often grouped with the untrainable feeble-minded or with the congenitally deaf.

A survey is made by Fumagalli of data in the literature regarding malformations and degenerations of the lower part of the labyrinth, in the hereditarily predisposed persons. The author points out that the examined cases may be divided into three great groups according to the period in which the hereditary factor exerts its influence, i. e., the morphogenetic period, the birth period, and the beginning of old age.

Arnvig subjected 486 children from the Danish state schools for deaf to a rotatory and a caloric vestibular test. The great majority of the children with inherited severe hearing loss had normal vestibular function. Abolished vestibular function in cases of sporadic recessive deafness was often combined with a coexistent or latent retinitis pigmentosa. The rule that the majority of cases with severe acquired hearing loss has abolished vestibular function holds only for hearing loss acquired after birth, whereas the reverse applies to those acquired in fetal life or during birth. The results of the vestibular tests showed surprising conformity for the group with inherited hearing loss and the group with hearing loss of unknown etiology. In 8% of all cases there was discordance between the results of the caloric and of the rotatory test.

Intracranial Pathology and Deafness

Eight case reports are given by Denny to illustrate the importance of a comprehensive examination for the early diagnosis of acoustic neuroma. The following routine is advocated in patients with acoustic symptoms: 1. Audiometry: either total deafness or a perceptive high tone loss are often typical;

recruitment is usually absent; loudness balance is of little assistance. 2. Corneal reflex: depression on the affected side is an early symptom. 3. Function of the facial nerve: paralysis is a late sign. 4. Spontaneous nystagmus: sustained rhythmic nystagmus, more marked to the affected side, is usually present. 5. Positional nystagmus: may be suppressed to the side of the lesion or in both directions. 6. Caloric reaction: the method of Fitzgerald and Hallpike frequently shows complete failure of response at 30 C and 44 C; complete loss of function with limited hearing loss is most significant. 7. X-ray tomography: can establish early diagnosis. 8. The Wassermann reaction: essential in every case of obscure perceptive deafness. 9. Lumbar puncture: a raised protein level in the C. S. F. is present in the majority of cases.

An accurate and detailed but mainly statistical study is made by Schulze of the anamestic data, of the subjective and objective symptoms, of the data supplied by neurological otovestibular, ocular, and laboratory examinations insofar as they are useful for an early diagnosis of tumor of the acoustic nerve. The material consisted of 91 cases of neurinoma of the acoustic nerve, 5 cases of meningioma, and 3 cases of cholesteatoma.

The symptoms of acoustic neurofibromata are described by Morris. Early symptoms, which are often relatively mild and therefore ignored or improperly evaluated, must be considered carefully. The tremendous importance of the vestibular responses and the frequent absence of x-ray evidence are stressed and must be appreciated for early diagnosis. The absence of x-ray evidence should not delay surgical treatment if the clinical findings indicate such a procedure.

The study of cochlear and vestibular function in seven cases of eighth nerve tumors, by Jatho, showed the limited value of audiological test in differentiating between medially and laterally situated tumors. An over-all hearing loss was commoner than an increasing loss in threshold audiograms. Absence of recruitment was found in only two-thirds of the patients. Partial recruitment points to lesion of Corti's organ from venous congestion.

Bocca et al. investigated the discrimination of distorted voice in 18 cases of supratentorial tumors by means of filtering the live voice through a low-pass filter. The results were significant, showing the score of distorted voice to be lower in the ears contralateral to the cortical lesion, while the pure-tone and speech audiometry did not show any deviation from normal on either side. Negative results with distorted voice were found only in cases where the lesion did not affect the cortical part of the temporal lobe. This loss of discrimination disappeared in three to four weeks after surgical removal of the cortical lesion. This improvement of the postoperative score for distorted voice seems, according to the authors, to support the view of bilateral cortical representation of each cochlea.

Tumors of the temporal lobe cause a directional preponderance toward the homolateral side; tumors in other regions of the brain do not give rise to this phenomenon. Especially the posterior part of the temporal lobe is thought, by Carmichael, Dix, and Hallpike, to be the source of the directional preponderance; mostly both caloric and optokinetic nystagmus show the same tendency. In some instances, however, only one of the two shows directional preponderance. On the basis of the finding, the authors give their theory about different centers and pathways for caloric and optokinetic nystagmus and other centers influencing their direction.

Twenty patients with clinically diagnosed arachnoiditis of the cerebellopontine angle were examined by Jatho. They showed a unilateral vestibular hyposensitivity without spontaneous central vestibular symptoms. Fourteen cases revealed progressive loss of hearing without compensation of the sound intensity. In the other cases, which, apart from the pain always observed in the back of the head, showed occasional seizures of dizziness, vomiting, and tinnitus, there was continuous loss of hearing, with positive

recruitment, as is also observed in Ménière's disease. The cause of the latter symptoms in such cases of arachnoiditis is therefore (from an audiological viewpoint) in the peripheral auditory organ.

Blagoveschenskaia looks at neural stimulation in the light of Vvedenski's theory of "parabiosis." According to this theory, under the influence of certain stimuli there may arise in the nerve a state of complete inhibition of conductivity, called by Vvedenski "parabiosis." This phase of inhibition may be preceded by two phases: (1) phase of uniformity, in which weak and strong stimuli give the same effect, and (2) paradoxical phase, when weak stimuli may elicit the response but strong stimuli remain without effect. Ageeva-Maikova showed a paradoxical reaction of the vestibular "analyser," in cases with a tumor of the posterior fossa, with use of the rotation test. In these cases, where rotatory stimulus was used in the normal way (10 turns) without effect, it was possible to elicit postrotational nystagmus by slow turning around two or three times. In the author's opinion, the caloric test with graduated stimuli (60 ml. of water at 25 C or 19 C) is better suited for observation of the phases of parabiosis. He often observed these phase phenomena in cases of tumors of the cerebellum or fourth ventricle before and after the operation. When calorization with a stronger stimulus (water of lower temperature) was performed, either duration or intensity of the nystagmus did not change (phase of uniformity) or reaction was markedly less (paradoxical phase), in comparison with the reaction to a weaker stimulus. The paradoxical phase could also be shown when examining for past pointing. In some cases, after a weak caloric stimulus past pointing was present, but it was conspicuously absent when stronger stimuli were used. In some cases of cerebellar tumors, mostly medulloblastoma, the author observed unusual graduation of responses to stimuli of different strength: e. g., no reaction to the ordinarily used caloric stimulus (60 ml. of water at 25 C) and a very intense reaction to a slightly stronger stimulus (60 ml. at 19 C). In some other cases he noted complete absence of the caloric reaction, whereas the postrotational nystagmus was stronger than usual. Some phasic disturbances of the rhythm of the caloric nystagmus were also observed, mainly in patients whose general condition was precarious. In these circumstances, when a stronger caloric stimulus was used, nystagmus movements became slower but more tonic undulatory. In the postoperative period, after removal of the tumor localized in the cerebellum or in the floor of the fourth ventricle, these phase phenomena often interchange, which seems to indicate the absence of gross morphologic changes and the presence of neurodynamic changes in the C. N. S. Complete inhibition of the vestibular reactions soon passes over into the paradoxical and uniform phase, which can usually be observed during 15-30 days after the operation. Gradually vestibular responses become adequate. With the deterioration of the patient's general condition the return of the paradoxical phase is often observed. Benign tumors of the posterior fossa usually cause paradoxical reactions only immediately after their removal; soon all phasic reactions disappear. On the other hand, in the cases of malignant tumors (or some very large benign tumors) the phasic responses, especially paradoxical reaction, may remain for a long time. In the acoustic neural apparatus phasic parabiotic responses are much more difficult to show. In the author's opinion, certain forms of hyperacusis occurring after removal of cerebellar tumors but also in other diseases of the C. N. S. are due to neurodynamic phase reactions of the acoustic "analyser." The circumstance that in some of these cases the whispering voice is relatively better heard than the spoken voice is a point in favor of this conception.

The case is reported, by Bonnefoy, of a girl, aged 19 years, who presented a tumor of the posteroinferior wall of the third ventricle, with nystagmus retractorius (rhythmic retraction of the eyeballs, 3-4 mm.

two or three times per second), associated with elevation paralysis. This association occurs habitually and may be completed by pupillary disturbances and retraction of the upper eyelid in cases of lesions of the Sylvian aqueduct. The symptom is of great significance for localization purposes; it is practically always the result of affection of the mesencephalon (region of the aqueduct, tectal tumor, affection of the cerebral peduncles of any nature). There is a supranuclear alteration near the posterior white commissure. The association with the Parinaud syndrome reveals its localization in the neighborhood of the vertical pathway. The prognosis depends exclusively on the nature of the lesion. Possibly, there is diffusion of the motor influx to all rectus muscles, which causes the retraction.

Medical Treatment of Deafness

Currotto Costa sets out the general indications and examines the pathogenesis and the lesions of certain kinds of deafness (conduction and mixed with intact drum). He describes in detail a method of perfusion through the drum by combining two injections into the ear cavity, hyaluronidase and a suspension of cortisone. He suggests Varidase, which acts well in the lysis of fibrin, leukocytes, and exudates. He also proposes heparin.

Forty-five hypoacoustic patients suffering from intractable tinnitus were treated by Caligaris with combined vitamin A and E. The best results were achieved in transmission or mixed hypoacousia. Encouraging results were also noticed in otosclerosis and in some forms of deafness of the perceptive kind.

Several authors have treated chronic progressive deafness and tinnitus with big doses of vitamin A, and the results thus achieved proved to be satisfactory in some forms of hypoacousia for which no recovery could be foreseen some years ago. Brunetti's inquiry considered 10 cases of labyrinthine hydrops, 10 cases of labyrinthine angioneurosis, and 7 cases of otospongiosis. In 70% of the first two kinds with auditory alterations, vessels proved to be very fragile, while in cases of otospongiosis the fragility positivity lowered to 57.1%. An endothelium protective therapy with vitamin E could improve vessel fragility as well as the subjective and objective phenomena of the disease.

Vitamin E, besides being an antithrombotic substance, can favor as well the forming of scar tissue, cell oxidation, and expansion and neoformation of capillary vessels. It has furthermore an antihyaluronidasic action and a control of capillary vessel permeability. An anatomopathoclinical opinion of "diffused disreactive mesenchymopathy" that can be applied so some peculiar diseases in otorhinolaryngology is expressed by Turrisi. As to the therapy, vitamin E is considered to have a very important action in preventing these diseases, as its action should work in these cases by entering the hyaluronic-hyaluronidasic acid system, which is very important in the fundamental building of connective tissue. Actually, in collagenosis the noticed alterations should be ascribed, when joined to E hypoavitaminosis, to insufficient antihyaluronidasic action, which helps depolymerize the mucopolysaccharides, and the consequent alterations in collagen. The most evident gains were noticed in patients suffering from hypacusis for a rather short time (10-12 months). On the contrary, when the process causing hypacusis is an old one evolving toward sclerosis, no gain could possibly be foreseen. The treatment with multiple vitamin A, E, and PP is advisable, with due care, for all those patients suffering for hypacusis of rather recent origin or in cases of perceptive hypacusia, when no organic lesion exists indifferent to the effect of an improved hematosis and more convenient humoral exchange between interstices and nervous sensorial cells.

Ferrari reports the results of administration in subjects over 65 years of age affected with senile hypacusia (presbyacusis) due to arteriosclerosis and hypertension. Ad-

ministration of nicotinic acid (Ronicol, Roche) exerts a specific action on the labyrinth. In cases of reversible changes of the neuroepithelium, one obtains now and then a transient improvement of auditory acuity.

Rossi reports that the combination of vitamin A, vitamin E, and nicotinic acid shows therapeutic efficiency in the treatment of some forms of chronic progressive hypacusia and of some syndrome having a vasculopathic vasomotor substratum of the posterior labyrinth. In the case of pure vertigo syndromes or of syndromes of angioneurotic substratum, this polyvitaminic association attenuated, both in intensity and quantity, the subjective symptomatology. Even when no gains were achieved (otospongiosis, tympanosclerosis) it prevented a further aggravation of the morbid symptoms, as noticed by inspection of the site after some months.

Experience in the treatment of subjective tinnitus in nerve hearing deficiency with Hydergin (a mixture of 0.1 mg. of dihydroergocornin, dihydroergocristin, and dihydroergocryptin) is described by Tanner. The treatment was based on the observation that the functional condition of the inner ear can be influenced by drugs acting on the A. N. S. Sympathicolytic drugs proved to be superior to parasympathicomimetic drugs as regards their effect on fatigue and recovery of hearing after sound stress. The report comprises 20 cases. In 12 of them, Hydergin therapy was successful. Four patients were completely rid of their tinnitus; the others improved considerably. No result was observed in eight. The investigations cover a period of three to nine months, and so no definite conclusions regarding the duration of the improvements can be drawn.

In a series of cases of perceptive deafness with tinnitus occurring in middle-aged patients, Pick assumes atherosclerosis to be the etiologic factor. The recent work on the biochemistry of atherosclerosis is discussed. The possible prevention of the condition is considered. Thirty-five patients with perceptive deafness were treated with heparin. There was no improvement in hearing, but there was some encouraging relief of tinnitus.

From 81 cases of mastoiditis, 31 were treated with x-ray by Brunar. Four types were observed: (1) recovery at once, (2) slow healing, (3) latent mastoiditis, and (4) ineffective treatment. The function of the treated ear was identical with that of the untreated one. The investigation was made between four months and four years after the treatment.

Somatopsychic and psychosomatic factors in tinnitus, deafness, and vertigo are discussed by Fowler and Fowler Jr. Somatopsychic implies that a patient is emotionally upset by his primary disease in some tissue or organ. Psychosomatic means that a real disease process has been set in motion by an overloading and breaking down of body defense mechanisms. Of adult patients, 85% complained of or have been conscious of head noises of some kind or other. The prime requisite for successful treatment is to explain in simple words the reason for the symptoms and then to make the patient understand how severe they really are. Proper medication is of service but will not suffice alone and may even produce undesirable consequences. A careful examination is necessary: detailed history, family history, general medical examination, measurement of hearing capacity, loudness and frequency of tinnitus, and estimation of degrees of vertigo, nausea, vomiting, and spontaneous or positional nystagmus. The results of the examination should be told to the patient in a confident reassuring manner, and the basic types of tinnitus and their pathophysiology should be explained simply and briefly. Impress on the patient that the symptom is an illusion of sound but not a delusion. It may be necessary to give the antihistamines, nicotinic acid, or any other drugs or even to advise operative procedures, but all of these patients need, as well, reassurance and insistence on developing a new philosophy of life.

Over 90% of 865 adults known to be using their hearing aids found them helpful, report Kodicek and Garrad. With advancing age the patients used their aids for shorter periods and on fewer occasions. The main criticisms of the aid were that it was cumbersome and got in the way at work, that the leads broke too often, and that reception was too noisy. The provision of an efficient hearing aid is the first step in the management of deafness. Lipreading, auditory training, speech therapy, and sympathetic handling of the various social and psychological problems encountered among these patients are needed.

To make a hearing aid more wearable and inconspicuous, with elimination of lost power and extraneous noise, a perforation is made by McDonald and Bach in the floor of the concha of the auricle behind the antitragus. Posteriorly it emerges at the lower end of the postauricular sulcus, behind the lobule of the ear. A one-piece ear mold, including the external meatus and the new aperture, is made. The lower end of the mold, behind the lobule, has a female opening which receives the male fitting, attached to the tube of the receiver.

Miscellaneous Papers on Deafness and Tinnitus

Observations on 1,857 persons who were subjected to the Nagasaki atomic bombardment were recorded by Goto and Murashima. These had experienced the bombing in 1945. They were located from 1 to 2 km. from the hypocenter. 1. Of these, 91 had residues of injury in the ear, nose, and throat. Most of these cases also had cataract as well as keloid formation on some other part of the body. No otolaryngological disturbances were found in those who had stayed at 3 km. or farther from the hypocenter. 2. In 36 cases, there was atrophic deformity of the external ear due to burns and perichondritis. 3. Seven had a perforated tympanic membrane; thirteen showed scars in the eardrum, and five had developed chronic suppurative otitis media. 4. Hearing impairment was found in 19, all suffering from conductive deafness. 5. No inner ear disturbance was noted. 6. Nasal injuries were extremely rare. 7. Nine had cicatrix in mouth and pharynx. 8. One case was fitted with tracheal tube on account of laryngeal stenosis caused by chondritis.

An audiometric study was carried out in 25 hypertensive subjects, by Di Nicola, of whom 15 had essential hypertension and 5 had arteriosclerotic and 5 renal hypertension. All of the patients showed the presence of tinnitus, of medium intensity, varying between 3,000 and 4,000 cps. In the three varieties of hypertension, there was hypacusia affecting the high tones of the auditory range, with an audiogram of perceptive type. The most marked modifications were encountered in the group of arteriosclerotic patients because of more marked and earlier damage of the specific receptors of the inner ear, which manifests itself precisely with an acoustic defect of perceptive type.

The differential elements between senile osteoporosis and Paget's disease at the level of the osseous labyrinthine capsule are reported by del Bo. Osteoporosis consists mostly of deficiency of the physiologic neoformation of the bone, resulting in the formation of fairly large medullary spaces filled with adipose or hemopoietic medulla, while Paget's disease is characterized by an osseous absorption accompanied by a neoformation of this tissue according to the different phases of the disease. The resulting medullary spaces are filled with conjunctive tissue rich in osteoblastic or osteoclastic cells. The differential histology of these two affections should be based on the elements that form the medulla and on the quantity of neoformed bone.

The eye and ear are close together in early embryonic periods, writes Neuberger. Developmental disturbances of the two skull blastems will produce malformations. A combination of this kind is retinitis pigmentosa combined with inner ear deafness and inhibition of pneumatization of the sinuses and mastoids. Retinitis pigmentosa

is an embryonic disturbance, always combined with other malformations. The frontals develop in the fourth embryonic month and are most prone to malformations, being the indication of the pneumatization pattern of the nasal sinus system. In the middle ear system the weakest points are the mastoid cells, which develop last and are most easily disturbed. We have never seen hypo- or aplasia of the Eustachian tubes or tympanum or antrum. The loss of function of the cochlear nerve represents an inner ear deafness with typical loss in the high frequencies. The neuroepithelium of the organ of Corti is believed to be disturbed in early embryonic life. The neuroepithelium for the high frequencies seems to develop later than for the middle and low frequencies. According to its developmental youth, it is less resistant to embryonic disturbances and strain during its lifetime.

The vestibular findings before and after operation in a case of platybasia with probable Arnold-Chiari syndrome are reported by Wodak. Prominent features are the complete absence of vertigo and nausea and the striking discrepancy between the strong nystagmus reaction and the normal deiterospinal reflexes.

Ricci and Ancetti have examined the structure of the temporal bone and of the aural structures in a case of gargoylism. They have noticed a particular compactness of the periotic bone structures and the presence among them of cartilaginous remnants. The anterior and posterior labyrinth appear to be normal. Lesions of degenerative thesaurismotic type have been found in the nervous ganglia. The lesions of the middle ear are limited to a modest thickening of the connective tissue ligaments. The personal findings are compared with those referred to by the literature on general lesions due to gargoylism and on alterations of the hearing apparatus.

A condition of thyrotoxicosis was induced in rabbits by administration of thyroxin for a suitable period, and the effects of this condition were studied in the inner ear. Marked degenerative lesions were encountered in the olfactory mucosa and in the inner ear. The greatest damage was done to the functional elements of the sensory mechanism—in the inner ear Corti's ganglion cells and in the olfactory system either the peripheral neurons or the bulbar fibers and the mitral cells themselves. Attention is called by Salerno to the possibility of these anatomical formations being involved also in cases of hyperthyroid syndromes.

The review of 8,000 cases of diabetes mellitus was undertaken by Kindler to determine the relationship of diabetes and deafness. Various authors have indicated that there is an incidence of 40%. Careful evaluation with audiometric studies in this group has shown that there is an associated arteriosclerosis, which is divided into approximately 36% among men and 45.8% among women. The type of deafness is invariably the perceptive type, although it may be mixed. The author believes that the evaluation of this study has failed to show a single case of deafness caused by diabetes. On the other hand, he believes that when deafness does occur in these patients it is due to arteriosclerosis.

With reference to a decision taken by an insurance company, the problem of the essential characteristics of hardness of hearing due to dystrophy (starvation) is discussed by Glatzel. The conclusion is reached that corresponding disturbances occur but that these are reversible injuries.

Anatomical and functional abnormalities in nose and epipharynx of aviators almost always lead to disturbances in the tubal function with repercussions on the elements of the middle ear, reports Robert. The barotraumatically caused disturbances in sound conduction develop in four stages: (1) the normal condition, with occasionally acute disturbances which heal completely or nearly completely. In many cases progressive lasting impairment is absent even after more than 10,000 flying hours; (2) intermittent tubal catarrh with sometimes mild, sometimes more marked lasting impairment.

In this stage treatment may still have a full effect; (3) continuous tubal catarrh; insufflation of the tubes, if possible, can be done only by catheterization. The impairment of hearing is always severe; (4) complete and definite tubal occlusion with fibrosis of the middle ear elements.

Sudden deafness may be the result of numerous different causes, according to Sterkers. Apart from the cases of transmission deafness which are evident at otoscopy, the other forms of perceptive type may be provoked by (1) infections (syphilis, epidemic parotitis, herpes zoster, encephalitis due to varicella, measles, etc.), (2) toxic agents (streptomycin, especially dihydrostreptomycin, and sometimes also neomycin), (3) tumors (acoustic nerve, pontocerebellar angle), and (4) trauma (cranial trauma, sound trauma). Finally, there are the cases of essential sudden deafness (virus neuritis, localized central lesions, arthritis, thrombosis, cochlear syndromes with hemorrhages or edema). Treatment must be based on etiology and pathogenesis and be given as soon as possible so as to decrease the duration of cochlear affection, which may be followed by permanent atrophy of the cells of the organ of Corti.

Klippel-Feil's syndrome, one of the developmental anomalies, is characterized by the bony fusion of cervical vertebrae. Three cases of this syndrome with complete deafness or bilateral, unimportant islands of hearing are reported by Bardadin and Siedlanowska. Vestibular responses were either completely absent or very weak on both sides. X-rays showed changes in the petrous bone (assymetry of pyramids) and in the structure of the inner ear. Thus, it is to be accepted that deafness in these cases is due to the malformation of the inner ears.

Emilio et al. present a discussion of hemotympanum, or blue tympanic membrane. They think that perhaps this condition is produced by an obstruction to the Eustachian tube, with a marked hyperemia exvacuo in the tympanic cavity, which produces a hemorrhagic condition. Paracentesis may have to be repeated with Eustachian tube inflation. The authors present a case in which, although recurrences did occur, they were finally able to effect a complete cure with repeated paracentesis and inflation.

An attempt was made by Friedmann to reproduce experimentally the clinical aspects of chronic purulent otitis media, and a study was made of the anatomical and clinical conditions which may favor the formation of cholesteatoma. Both in experimentally affected guinea pigs and in cases of acute otitis and of chronic otitis the author observed the existence of active osteogenic processes, leading to total or partial obliteration of the preexistent pneumatic cavities. The chronic cases showed, furthermore, marked processes of epithelial metaplasia in the mucosa, with formation of zones covered with epithelium of the respiratory type, containing numerous glandular formations. Many cases showed the presence of cholesteatomatous formations, although without cholesterol deposits. The cholesteatoma is formed by penetration into the tympanic bulla of the guinea pig of multistratified pavement epithelium, deriving from the tympanic membrane or from the external auditory duct.

Schuknecht has made an interesting and quantitative study of the pathological anatomy in the ears of several cats and humans with high-tone deafness. The evidence indicates that there are two types of presbycusis. The first, termed epithelial atrophy, is characterized by degenerative changes which begin at the basal end of the cochlear duct and proceed toward the apex. The second type, neural atrophy, is characterized by degeneration of spiral ganglion cells, beginning at the basal end of the cochlea, as well as neurons of the higher auditory pathways.

Simonton states that treatment will not prevent or reverse the loss of hearing with age. Careful enunciation by the speaker, avoidance of confusing noise, speechreading, and the use of a hearing aid all help the

presbycutic. The limitations of amplified speech in presbycusis must be realized to achieve satisfaction with a hearing aid. Retraining of listening habits requires up to six months of persistent and conscientious practice with the hearing aid. The following factors favor hearing of conversation: (1) complete enunciation, that is, provision of a complete sound picture at the source; (2) slow rate of speech, allowing time to fill gaps in the sound picture so that the listener may understand the sentence although he misses some words or syllables; (3) facing the listener when speaking, which brings the maximal sound pressure to the listener's ear; (4) avoidance of interfering noise when speaking, and (5) speaking at normal loudness; shouting encourages poor enunciation.

Bordley and Haskins consider a group of older patients who exhibit subjective (standard test technique) audiograms, indicative of good ability to wear a hearing aid, but who prove to be unable to profit from amplification. It is reported that persons in this group commonly have higher PGSR than standard subjective audiograms, that thresholds for awareness and understandability are notably disparate, and that understanding grows markedly worse when speed of delivery is increased. Reasoning from certain conditioning experiments on decerebrate animals, the authors conclude that the clinical and laboratory observations, considered together, suggest that the difference between the hearing thresholds by subjective and objective audiometry indicates approximately the degree of cerebral sclerosis of the auditory area.

The clinical evaluation of presbycusis, on the basis of different tests of auditory function, was carried out by Pestalozza and Shore in a group of 185 selected old persons. The relationships between different tests of auditory function gave the following results: 1. In old persons, discrimination for speech is very poor, even in the presence of a mild hearing loss. A parallel investigation in a smaller group of young persons with the same amount and characteristics of hearing loss as the old ones showed that young ones have better discrimination. 2. In old persons, a clear relation exists between hearing loss and discrimination loss; discrimination becomes poorer as hearing loss becomes greater. 3. No significant relation has been found between slope of the audiogram and discrimination loss. 4. The relation between hearing loss and discrimination loss in a subgroup of persons with positive medical history does not differ significantly from the relation in the total group of old persons. 5. The relation between hearing loss for pure tones, hearing loss for spondees, slope of the audiogram, and discrimination loss shows that persons with a steep slope of the audiogram (more than 20 db. per octave) have a smaller hearing loss for spondees than can be predicted from the audiogram (average 500-2,000 cps) with respect to persons with almost flat or mildly sloping curves. Furthermore, in persons with good discrimination, hearing loss for spondees is a little less than for pure tones (average 500-2,000), whereas, in persons with poor discrimination, hearing loss for spondees is definitely greater than for pure tones. 6. Since in a group of young persons the agreement between hearing loss for pure tones and for spondees is much more precise, these results substantiate the hypothesis that in old persons a "phonemic regression" syndrome exists. Phonemic regression seems to have a nonperipheral basis. 7. In a smaller group of 24 old persons on whom a recruitment test was performed, 50% of the subjects showed no recruitment, 30% partial recruitment, and 20% almost complete recruitment. The absence of recruitment in most of our cases of presbycusis is in good agreement with those histological findings that indicate spiral ganglion cells and nerve fibers as the commonest lesions responsible for hearing loss in presbycusis.

Factors in the differential diagnosis of retrolabyrinthine hearing disorders are discussed by Wildhagen. In unilateral hearing disorders the following factors are important for the diagnosis of disseminated encephalomyelitis: (1) the sudden onset of a deaf-

ness, with tinnitus but without vertigo; (2) the audiologic proof of a retrolabyrinthine nerve stem disturbance, with negative recruitment; (3) peripheral underexcitability or inexcitability of the vestibularis, with preserved pointing reaction; (4) possible occurrence of positional spontaneous nystagmus; (5) negative finding in Stenger's test and skull x-rays, and (6) cytoalbuminocolloidal disassociation in the spinal fluid. Repeated otological and neurologic examinations are necessary, since the findings are variable in disseminated encephalomyelitis.

Retrolabyrinthine deafness is discussed by Krassnig. Brain stem deafness is of increasing interest in dull skull injuries, since damage of the cochlea may be the only permanent symptom. A total of 20 cases of encephalitis, vascular disturbances, and skull trauma of the brain stem were seen. In encephalitis bilateral deafness, especially for the low frequencies, and tinnitus were the first symptoms and were interpreted at first as otosclerosis. Later on, further bulbar symptoms explained the pathogenesis. The histologic findings were multiple small fresh and healed inflammations in the rhomboid fossa and pons. One case of brain stem damage of vascular origin with marked deafness, speech disorder, and pyramidal disturbance developed during surgery and was believed due to fat embolism and a patent foramen ovalis. Bilateral disturbance of hearing and speech permits the diagnosis of brain stem deafness with supranuclear location. The deafness and speech and tinnitus disorders in spotted fever are due to an encephalitis of the pons.

This new series, by Pagano, of researches in barotrauma by Caisson disease has been carried out on 104 patients, and, in comparison with the results obtained in the first experiments, has manifested an increase in perceptive deafness (76.2%) together with a decrease of conductive (16.5%) and mixed (9.8%) deafness. In perceptive deafness, to a percentage of 24.6%, the auditory lesion presents clear characters of retrocochlear localization.

Vestibular function has been investigated in 25 cases of otitic barotrauma by Caisson disease chosen from the cases already audiologically studied. After a short review of the pathogenesis of the barotrauma and the pathological findings in these cases, Pagano, on account of the peculiarities of the auditory and vertiginous syndromes, comes to the conclusion that in such patients the vertigo is often to be considered of extralabyrinthine origin.

A description is given by Chiarino of a patient with tinnitus of the left ear of pulsatile type, synchronous with the pulse, which lasted for two years. Arteriography revealed the existence of a malformation of the external carotid. Ligation of the occipital artery, blocking of the left sympathetic, and treatment with Phenergan gave no or only transient improvement.

Timofeyeff, Tolokonnikoff, and Pokrivalova discussed the sound spectrum of tinnitus and the threshold of covering of the tinnitus. They found that some sorts of tinnitus consist of one or two or more tones. The threshold of masking was 70-80 db. or more, mostly of a frequency of 4,000-6,000 cps. In some cases tinnitus disappeared after cessation of the covering sound for one to three minutes. The authors explained this occurrence as the result of depression of the hemisphere.

Tinnitus may be cured by injection of Impletol, reports Brisotto. The mechanism of tinnitus is obscure. Impletol is a mixture of equal parts of procaine and caffeine and is injected subcutaneously in the mastoid region. Little is known of its mechanism of action, but it must act on the diencephalic centers.

Homolateral hearing disorders in amputees are reviewed by Schnurbusch. Large scars act as irritation centers and set up vasomotor, sensory, or dystrophic disturbances in other parts of the body by an unphysiologic reaction of the vascular system and the accompanying autonomic system. The projection to another part of the body is usually homolateral. Homolateral vascular disturbances of the retina are seen

in amputation scars of the limbs. Sympathetic disturbances of the eye are associated with chronic otitis media or radical mastoidectomy. Of 26 patients with limb amputation examined, 10 had aural disturbances. This was explained as follows: By means of a reflex arch through the periarterial nerve fibers and the sympathetic chain, a vascular disturbance in the inner ear is followed by pressure changes, setting up a functional disturbance.

Intensive unilateral perceptive deafness of sudden onset without vestibular disturbance may be reversible if seen early. It is due to cervical spine changes, disturbing the sympathetic innervation to the ear. Bieckert states that Glisson suspension and stellate blockage with 1% Novocain may restore function completely. In unilateral perception deafness of unknown origin the cervical spine should always be checked.

Cervical segmental therapy for inner ear disorders is presented by Lanzendörfer and Fickentscher. Segmental therapy means utilization of the cutivisceral reflexes. The upper cervical segments are predisposed developmentally for therapeutic applications, since hearing plate and hearing vesicle arise from the skull epidermis. The therapy has an effect direct to the inner ear, and the segments C2+C3 offer an indirect effect through the sympathetic system of the labyrinth ascending along the vertebral artery. Normalization of vascular disturbances and labyrinth hydrops may be achieved this way. They used Dodecatol-Heyl (Cetain, nicotinic acid, and cyanocobalamin). Every second day 2 cc. of Dodecatol is injected subcutaneously in the segments C2+C3 bilateral of the spinal processes. The course includes six injections. A repetition after four weeks is indicated in every case. Of 24 patients with different forms of inner ear deafness, 7 had improvement of more than 20 db. in 500 to 2,000 frequencies. Nine patients improved over 20 db. in the 4,000 frequency and less in the main speech frequencies. Eight patients reacted unsatisfactorily. All patients reported a marked decrease of the tinnitus and a hearing improvement greater than really found in the audiogram.

Therapeutic puncture of the vertebral artery in hearing disorders is reported by Mayer. With the development of arteriography intra-arterial injections were done to dilate vessels, which were closed by spastic, thrombotic, or embolic conditions. The best dilatating effect was seen with acetylcholine. The arterial injection permits a higher concentration of the medicament in a certain area, which is not possible by the intravenous route. Fewer injections are necessary, often only one. In functional (spasm) or organic (thromboendangiitis oblit) changes of the blood vessels of the inner ear, the vertebral artery is punctured and Ronicol injected. At the same time the plexus surrounding the artery is blocked with Procaine. In this way the vasodilatation is prolonged. Patients with a sudden deafness and some with prolonged tinnitus were treated this way. In one case, normal hearing was noted after one injection. A second case showed marked improvement. Tinnitus showed improvement six times in 14 patients. Up to five injections every second day or at weekly intervals were given. Technique of puncture: The needle is inserted at the upper end of the thyroid cartilage paramedially. The carotid artery is pushed to the side; the needle is brought in opposition to the transverse process of the spine and then pushed forward one more centimeter, where the vertebral artery and the plexus are located. Puncture of the dural sac has to be avoided; aspiration is necessary. The injection has to be done slowly to avoid nystagmus. The nervus vestibularis is influenced as well. By means of nystagmograms it is shown that the nystagmus is more frequent and of lower amplitude after injection of Ronicol. The verebral artery is chosen for injection, since the arteria auditiva interna comes from the basilar artery or seldom from the inferior cerebellar artery. Injection into the carotid artery would not reach the inner ear.

The first part of the communication by Motta and Profazio contains a review of

the controversial problems and the views of earlier authors on the pathogenesis of vestibular and cochlear symptoms arising as the consequence of lesions of the posterior cervical sympathetic (vertebral nerve and perivertebral plexuses) or of the cervical sympathetic ganglion chain and of the pericarotid plexus; further, there is a discussion of the clinical observations and earlier experiments on the relations between cervical sympathetic and the labyrinth or the nuclei and the central pathways of vestibulum and cochlea. Subsequently, a report is presented on a clinical case in which rotation of the head to the right was followed by attacks of vertigo and other subjective symptoms, associated with nystagmus, right auditory deficiency, vasoconstriction of the cephalic territory (recorded by photoplethysmography at the lobule of the ear), tachycardia (demonstrated by ECG), and increase of blood pressure measured in the upper arm (studied by oscillometry) and pressure of the central artery of the right retina, with mydriasis. On the basis of the observation that these symptoms temporarily disappeared after administration of drugs with ganglioplegic action and after procainization of the right cervical sympathetic, and of an accurate analysis of the various symptoms, the authors assert that the syndrome shown by this patient is the consequence of abnormal stimulation of the right cervical sympathetic ganglion chain and that the cochleovestibular disturbances are due to alteration of the functional condition of the peripheral receptors. The authors finally discuss the difference that exists between the syndrome observed by them and the classical syndrome of Barre-Lieou.

Continuous headache with paroxysms, dizziness, tinnitus, and eye complaints are the main symptoms of Barre's syndrome. The disease is caused by cervical rheumatic deviations which should irritate the sympathetic nerve. Therefore its name "syndrome sympathique cervical posterieur." X-ray examination of the neck is necessary to make the diagnosis, and x-ray irradiation of the cervical vertebrae gave the best therapeutic results, according to Muller.

Disease of the neck is a not uncommon cause of vertigo in man; Ryan and Cope suggest that this syndrome be called cervical vertigo. The authors have so far found three groups of patients who have this syndrome; (1) patients with cervical spondylosis, (2) patients treated by neck traction, and (3) patients with certain types of neck injury. Such patients usually have pain in the back of the neck and occipital regions, sometimes accompanied by stiffness of the neck. After a varying latent interval, recurrent postural vertigo, occurring on lying down and on bending the neck into certain positions, is noticed. This symptom may be reproduced and nystagmus induced by bending the neck but not by merely positioning the head in space. The prognosis is good, and relief may be obtained by wearing a cervical collar until the underlying lesion has resolved.

(To be continued)

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Jack Allan Weiss, M.D., President
Fletcher Austin, M.D., Secretary
Monthly Meeting, Dec. 1, 1958

Cancer of the Head and Neck: Primary and Reconstructive Surgery. Dr. Richard T. Farrior (by invitation).

The alert otolaryngologist, by nature of the physical examination of his patient, should detect more primary neoplasms of the head and neck than any other specialist or general group. The otolaryngologist should acquaint himself with management of these tumors, whether or not he, himself, does the major surgery. If he is to understand the treatment of these lesions, whether large or small, he should be familiar with the methods of reconstruction available, as either primary or secondary procedures. A thorough knowledge of reconstructive aspects of this surgery allows the surgeon to be more radical in his initial excision or prevents him from being too timid, thus jeopardizing the possibility of cure.

Primary reconstruction, where there would be a significant disturbance in function without its use, has much in its favor. Statistics show that the time of the initial surgery is the best opportunity for cure. Also, with some form of primary reconstruction to allow more nearly normal function, even if there is a recurrence, the patient's chances for cure are reduced, but, in the meantime, he has had a more comfortable and happier life. Decreasing the severity of the deformity often aids in maintaining the integrity of the patient's personality. Additional curative surgery can still be performed.

Color transparencies, representing common and unusual lesions from each of the anatomical areas of the head and neck were presented. Photographs made during surgery, representing particular techniques, extending from simple skin lesions to major en bloc combined neck resections were presented.

Conclusion.—A plea is made for early detection and adequate primary excision, which should make this extensive surgery unnecessary and insure a much higher cure rate.

Unnecessary discomfort and disfigurement in this area of eating and breathing can often be prevented by some form of primary or secondary reconstruction. If reconstruction in any way would alter the main objective of curing the cancer, it should not be given any consideration.

Discussion

Dr. Maurice F. Snitman: Mr. Chairman, ladies and gentlemen, this evening's presentation is most commendable. Dr. Farrior is to be highly complimented on the excellence of his reconstruction procedures.

I wish to comment only on those procedures of partial laryngectomies where, in general, a total extirpation of the larynx is the general rule. I refer not only to some of the cases present here this evening but also to supraglottic and hemilaryngectomies. Those procedures find favor in some hands, but the possibility of inadequate resection can occur in not a small percentage of cases. I concur with my colleagues that in surgery of malignancy of the larynx limiting procedures are to be seriously questioned, when total laryngeal removal is much less likely to result in local persistence of disease.

Dr. Paul H. Holinger: This was certainly a comprehensive presentation. One of the points I would question is the need for radical neck dissection in all cases in which the cord is fixed. Did the essayist mean that neck dissection would be done simply because of fixation of the cord? Is there no qualification as to extent of lesion? In other words, if a lesion is limited to the cord and does not extend subglottically or supraglottically, does Dr. Farrior advise a neck dissection merely because the cord is fixed? I emphatically agree with Dr. Farrior that there is no reason that so-called neck surgeons should assume control of laryngeal surgery. Laryngologists have the ability and know-how to select procedures which will preserve function and still show as good or even better statistics in the management of laryngeal cancer. They realize the significance of cancer, but the experienced laryngologist, such as Dr. Farrior, often can, with ample justification, tailor the procedure for conservation of function.

Dr. Burton J. Soboroff: There is little for me to say except that Dr. Farrior's presentation was excellent and covered as wide a range as could possibly be covered in a short time.

There have been several questions about which some difference of opinion has occurred within our own department regarding the management of certain of these tumors. I should like to ask Dr. Farrior to answer several questions in view of his experience. Several cases of carcinoma of the ear were shown. The question is relative to those with the auricle involved, where cartilage which is in the region of the concha is affected. Is it better to do a total excision of the auricle than a partial excision? Dr. Farrior's results were beautiful in those in which partial resection was done, when the cartilage underneath was excised and healing and cure were effected. However, there are those in our group who believe that in such instances the entire auricle should be removed because of the grave prognosis.

The second question is in regard to the use of bone graft in mandible resection. I do not recall that bone grafts were used in the cases presented, but a pin was shown. Do you use bone grafts, and are they used primarily or in secondary reconstruction?

The third question refers to intranasal and antral tumors, such as inverting papillomas, in which very often there is much more serious involvement. There are some who feel the palate can be preserved by performing a wide resection through a Weber-Ferguson incision if the tumor is chiefly in the middle meatal region. Others feel the palate should routinely be removed. I should like to ask Dr. Farrior's opinion on this matter.

Dr. E. M. Skolnik: I wish to ask Dr. Farrior whether there is a problem feeding patients who have had the transhyoid procedure? How long is the tracheotomy tube kept in place, particularly when the epiglottis is removed?

Dr. Thomas Galloway: This was a beautiful and courageous presentation. If there was any question it was that we had no correlation of results with techniques. Final judgment must wait for such correlation. I do not know whether Dr. Farrior has been doing these procedures long enough to have five-year cures. I think as surgeons become more competent and do this sort of surgery, they may become bolder and develop procedures which only later may prove to be justified.

Peterson cited a report of over 100 cases, at the Curie Institute, of carcinoma of the floor of the mouth in which the procedures were very much as outlined here. However, he was very disappointed with the results of the radical procedure. I should question how often it might be advisable or necessary to do a radical procedure for a tumor of the mandible. At the Curie Institute they had very excellent results with intensive irradiation and concluded that more formidable surgery was not justified. Block neck dissection is advisable in any extensive involvement, even in the absence of palpable nodes. I should think in a carcinoma with overlying perichondrial involvement that the larynx should be removed. We have also found that carcinoma of the auricle is a treacherous lesion. Certainly in some cure is only possible with resection of the auricle; some may be treated by irradiation, and some may be treated satisfactorily with a minimum of surgery and diathermy.

Dr. Richard T. Farrior (by invitation): I very much appreciate this discussion and the exchange of ideas. I am sure I shall go away having learned a great deal.

I should like to make a few comments in answer to some of the points brought out in the discussion.

Dr. Snitman, I very, very much do not believe in partial excision of any carcinoma. The partial procedures outlined are those for more adequate wide-field local excision, where local excision is indicated. I should advocate partial procedures for laryngeal carcinoma where the very clear-cut indications for laryngectomy are not found. I rarely do the skeletonized laryngectomy but much prefer the wide-field laryngectomy with dissection to the carotid artery bilaterally, including the pretracheal tissues. Where the specific lesion does not contraindicate, I believe strongly that all tissue possible and, more important, all function possible should be preserved.

As far as the suprahyoid lesions are concerned, again they may be handled by a wide local excision. The indications for laryngectomy are fairly clear-cut, as is well known to most of you.

In transhyoid dissections, there is a great deal of trouble feeding the patient for some time. The length of time the tracheotomy tube is left in place depends on the individual case. We have less trouble by leaving them stoppered with a finger cot and sponge, as described in the presentation.

As far as fixation of the vocal cord is concerned, this is the least lesion for which we do a neck resection. With fixation in an originally intrinsic carcinoma, the lesion microscopically has not remained intrinsic. That is the thinking at the present time.

I believe in our discussion we are all going back to, and are concerned with, parital excisions. It is strictly a matter of individualization. I believe we are open to clinical criticism unless the type of lesion is correlated with the degree of individualization as far as the pathology is concerned.

In answer to the question about removing the ear for a carcinoma, I do not believe that simply because any part of the ear is involved the whole ear should be removed. Again judgment for the particular lesion is essential.

We have used bone grafts following mandibulectomy; however, I am very strongly opposed to using them primarily. They have been used where I had my training, and almost invariably in their hands and in mine they were lost when used primarily. Sometimes we wait until we put a secondary skin graft inside of the mouth before inserting the bone graft. I do not advocate primary bone grafts then, after mandibulectomies. I use the crest of the ileum as much as I can, and sometimes the rib.

In cases of inverting papilloma of the nose, I believe you can preserve the palate in many instances. In these areas the three-dimensional excision and thinking is essential. Again I think that it is most important to obtain adequate exposure for excision in all dimensions. To save time I edited my material, which illustrates one case where we excised the superior table of the palate for an adequate margin. This was a malignant melanoma of the lateral wall of the nose, but since it was not close to the palate, the inferior table was preserved.

Again may I say that this material presented an over-all coverage of the subject without being able to cover all the pros and cons. In answer to one member's question regarding my age, I have my five-year cures, but I draw very heavily on the experience of my seniors, some of them in this room. I do not alter my approach one iota from accepted principles of cancer surgery.

What makes one decide to do a particular procedure? I have drawn heavily on a review of the literature and the teachings of my professors. Sometimes at a distance you can evaluate these procedures a little better. I believe we should all exchange ideas as we have done here tonight and that we will all have a little broader understanding of the whole problem and what the statistical results mean.

A final point. The type of procedure in each case will depend on your judgment, correlation of all of the factors, and definite individualization of each case. If reconstructive procedures raise any question regarding adequate initial excision, it should not be given any consideration.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Monthly Meeting, Jan. 5, 1959

Cerebrospinal Rhinorrhea: Case Report. DR. BURTON SOBOROFF.

A report of a 20-year-old white man with cerebrospinal rhinorrhea was presented. When first seen, the patient had a clear, colorless discharge from the left side of the nose, which was intermittent in character and became more profuse when he leaned forward. The fluid showed the same consistency as spinal fluid when tested. There was no history of trauma, and physical examination, chest x-ray, blood and spinal fluid tests, fundi, and cranial nerves were normal. X-rays of the sinuses in the Water's position showed some clouding of the left antrum, and a large, smooth discrete mass in the left frontal region. A tentative diagnosis of osteoma of the left frontal sinus was made.

Chemical determinations on fluid collected from the nose of the patient proved it to be cerebrospinal fluid. Several tests may be used for this purpose, namely, the sugar reduction, the protein content, and the injection of dye by means of lumbar puncture. Among the causes of cerebrospinal rhinorrhea, as outlined in the literature on the subject, are acute head injury; an extensive or difficult nasal operation; sinusitis with necrosis and break-through; tumor, such as an osteoma or pituitary tumor or meningioma, and prolonged increased intracranial pressure with demonstrable congestion or a meningocele or meningoencephalocele. If none of the above can be demonstrated, there may be spontaneous leakage of fluid. Such spontaneous leakage may take place if the openings in the cribriform plate are unusually large.

It was pointed out that meningitis is very frequently a complication of cerebrospinal rhinorrhea, and bouts of recurrent meningitis should make one consider the presence of cerebrospinal rhinorrhea.

The treatment of cerebrospinal rhinorrhea is, in part, dependent upon the site of leakage. In the presence of a frontal osteoma, the best approach is transfrontal. In 1933, Fox reported the use of 25% silver nitrate local application to the upper straits of the nose, with complete cessation of the leakage of cerebrospinal fluid. Usually the surgical treatment of this condition is the most satisfactory. Intracranial repair of the dural leak may be necessary.

In summary, cerebrospinal rhinorrhea is an unusual cause of nasal discharge. Such discharge occurs most frequently after trauma. Meningitis makes it imperative that the site of such leakage be closed either by local treatment or by surgery.

Discussion

DR. LUIS V. AMADOR (by invitation): Mr. Chairman, members, and guests, it is a pleasure to come here as a guest and hear this very fine presentation.

Cerebrospinal rhinorrhea is certainly one of the most difficult topics with which we have to deal. I should like to comment on several things which are of interest from the neurosurgical standpoint.

Among the tests to aid in the diagnosis of cerebrospinal rhinorrhea, we have found the postural test most helpful. When the patient sits up, the leakage stops immediately. Since cerebrospinal fluid does not contain mucus, the patient's handkerchief may be checked for the type of secretion. If there is mucus, the cloth will become stiff upon drying. Very often there is anosmia on one or both sides.

The majority of the cases I have handled in the past, roughly about a dozen, were of traumatic origin. One little boy fell three stories from an open window and had traumatic cerebrospinal rhinorrhea with repeated bouts of meningitis. I would like to suggest that everyone who sees these patients be aware of this as a possibility.

In trying to determine what caused the leak in your patient, I find myself impressed by two possibilities. One is an encephalocele, a congenital defect, which appears in the midline. About three years ago we had a youngster who had a similar congenital encephalocele with a marked pulsation. However, it would be unusual to see an encephalocele arise in this case. It might have pressed into one sinus more than the other. The other thing is that, although this patient denied a history of recent trauma to the head, perhaps he had had an old skull injury which he did not recall. Sometimes a small fracture pinches the dura and the pulsation of the brain may cause a meningocele.

As all of us have seen, of course, many of these rhinorrheas clear up spontaneously. We are fortunate to have the antibiotics.

As far as surgical correction of cerebrospinal rhinorrhea is concerned, some cases can be very difficult. Whenever such leakage comes from the cribriform or from erosion of the pituitary, the procedure is complicated. Often we inject indigo carmine or methylene blue by lumbar puncture as we do a transfrontal craniotomy in order to see the dye and find the exact source of the leakage.

The slide shows several things which are of interest. First is the traditional fracture through the frontal sinus accompanied by leakage of cerebrospinal fluid with a pneumocephalus. The transfrontal approach has proved the most satisfactory in our hands, but unfortunately in such cases we have had a resulting anosmia which has been permanent.

The only satisfactory way to seal off such a defect is with fascia lata, or fascia from over the temporal bone. Packing and coagulation is the final step in sealing off such a defect.

In cerebrospinal rhinorrhea we have a condition which is a real challenge in the diagnosis and treatment.

I have enjoyed very much having the opportunity to be your guest tonight.

DR. FRANCIS LEDERER: I have several points which I thought might be worth mentioning. In the first place, in a patient first judged to have a vasomotor rhinitis, it might be well to say that meningiomas do not come to us as clearly represented as the essayist has indicated. Secondly, the rounded shadow we saw in this man's frontal sinus was the source of a good deal of conjecture at our seminar, and no one had the correct view of what the case ultimately proved to be at surgery. What presented in this man's frontal sinus might just as well have appeared in the nose, or, I might mention for the sake of the younger men, as a solitary polyp in the left side of the nose. What this man actually had was an encephalocele. It was in no way a tumor in the frontal sinus per se. Since we have these cases every fortnight (I am, of course, speaking facetiously), naturally we were well prepared at the time of surgery. When the pedicle came away, it was clear this was not a recent fracture but a dehiscent area of long standing. I thought these points might be of interest.

As to the method of handling these cases, I may say, Dr. Amador, we believe that the rhinological approach was easier than the neurosurgical approach would have been. We were directly down on the dehiscence, and it was obvious that we could manage it better and take the proper precautions. This man had a very nice result. I do not think we should fail to pay tribute to the antibiotics in these cases. In the preantibiotic era, Dr. Soboroff's case would have been rather hazardous, to say the least. We certainly would have had meningeal manifestations and some postoperative headaches.

Did Dr. Soboroff make any comment about whether 18 months is really sufficient time to guarantee that this man's dehiscence is well sealed over by scar tissue?

DR. MARK C. WHEELOCK (by invitation): It would have been of interest to have had a Gram stain. Ordinarily this fluid is clear and there is an absence of any bacteria.

DR. BERNARD COHEN: I am interested in this discussion because a few years ago we saw two such cases within an extremely short period of time. One was an extremely obese woman who was a bartender. She had had a nasal discharge for years which became very much worse after

violent sneezing attacks. We saw her through an allergist who tried to cure the increased nasal discharge previously by various skin tests and desensitization. The outcome of this case was successful by the neurosurgical approach, very much as Dr. Amador has described.

The second case was that of a young man who had had his fourth or fifth attack of meningitis. The first few were diagnosed as cerebrospinal meningitis and the subsequent attacks as pneumococcal meningitis. He recovered on antibiotics and we saw him accidentally with a very minor nasal discharge. He was tested and the discharge found to be a cerebrospinal rhinorrhea. X-rays were made and showed multiple small shots in the cribriform plate. When we rechecked his history, we found he had been exposed to buckshot some time previously. I do not know the outcome of this case because he was referred to a doctor out of the city.

DR. JACK A. WEISS: I wish to comment on the sugar-reducing test, either the Fehling or the Benedict.

D. Higbee, in an article in *Otolaryngology* (Coates, G. M., Schenck, H. P., and Miller, M. V.) related that in a specimen of cerebrospinal fluid examined 18 hours after collection from the nose, the test was negative, due to fermentation of the sugar.

The dextrose may be exhausted by metabolic action of the cellular or bacterial content of the fluid.

Strangely enough, two of the standard texts on clinical laboratory diagnosis fail to mention this pertinent fact. However, two biochemists whom I consulted confirm the statement, without specifying a definite number of hours.

To avoid being misled by a false-negative test for sugar in a nasal rhinorrhea suspected of being cerebrospinal fluid, it is most important to avoid an undue delay, such as would occur if a patient brought in a specimen collected at home.

DR. BURTON SOBOROFF: I should like to thank all of the discussors for their comments on this material. One of the things which was brought out was that occasionally sneezing brings on the sudden onset of cerebrospinal rhinorrhea, as mentioned in the literature.

Dr. Wheelock's comment about the Gram stain might be very worth while. One difficulty, however, is that the secretions might wash out cells or the bacteria from the nasal cavity.

In regard to the surgical approach, I can only say that the transfrontal one is much easier, and, if cure can be accomplished satisfactorily, might be a worth-while one. If not, then we must call in the neurosurgeon to give us help.

Thank you very much.

Extranodal Malignant Lymphomas. DR. MARK C. WHEELOCK (by invitation).

Approximately 200 cases are included in this series of extranodal malignant lymphomas. The primary concept is that the entities as such are first recognized and diagnosed in tissues other than lymph nodes (tonsils, spleen, and thymus, which are predominantly lymphoid in character) are also excluded. Nearly all tissues and organs in the body are included as primary sites. Many are in the head and neck region and hence will be of interest to the otolaryngologist. Although these will be stressed, there will be a quick summation of the others in the group. There will be a brief explanation and classification of the malignant lymphomas at the introduction.

Malignant Lymphomas of the Pharynx. DR. STANTON A. FRIEDBERG.

A series of 16 patients with malignant lymphoma of the pharynx was reviewed. Correlation of these tumors as to cell type is very difficult, and there is often a difference of opinion among pathologists as to diagnosis. They may be classified as malignant lymphoma, leukemia, Hodgkin's disease, lymphosarcoma, and giant-follicle types. An attempt was made to subdivide the type of tumor—for example, reticulum-cell sarcoma and lymphocytic sarcoma—in an effort to predict the prognosis. It would appear that there is some question as to the practical value of any such attempt at subdivision. There was a tremendous variation in the histological pattern in these cases, even in different areas of disease in the same patient. One area may show a reticulum-cell sarcoma, and another, Hodgkin's disease. This would speak for an inter-relationship between the various types. Many people believe that follicular lymphomas frequently eventually become reticulum-cell sarcomas and reticulum-cell sarcomas become Hodgkin's disease. These conditions in the pharynx are but local manifestations of a systemic condition that, in all probability, is uniformly fatal.

Of the 16 patients in the series, 14 have died. Two of the patients, who, from the histological point of view, should have died early, lived the longest. One patient, with a diagnosis of Hodgkin's disease in 1936, lived until 1948. Thus, the periods of remission may be very long, and, more than any other factor, offer some guide as to prognosis.

Discussion

DR. BURTON SOBOROFF: I should like to ask two questions and also express my appreciation of both papers, which were excellent.

Dr. Wheelock, was it suggested in your presentation that these are primary extranodal lymphomas, or may they simply be metastases with a primary elsewhere? Secondly, does the so-called

plasma-cell lymphoma fall into this group of lymphomas? One may also call them plasmacytomas. We have seen several of them in the nasal cavity.

DR. FREDERIC J. POLLOCK: I wonder if some of these might be leukemic infiltrates in these areas rather than primary lymphomas in the spot where Dr. Wheelock found them?

DR. PAUL H. HOLINGER: This has been a most stimulating evening. There are two things I wish to mention. Dr. Friedberg discussed these lesions as usually nonulcerative. I wonder whether he considers this as one of the diagnostic criteria or whether they are occasionally ulcerative? Our impression is that in the pharynx or larynx these lesions are not in any way characteristically either ulcerative or nonulcerative. We see just as many in the larynx which show ulceration; others are nodular, and some are nonulcerating as described. The second point is that in these lesions we are impressed by the fact that therapy is very effective for a long period of time, but then the disease breaks out everywhere and no type of therapy seems to be of benefit. I wonder whether this is the same impression Dr. Friedberg has had in his cases? We have found that patients who have responded beautifully to their initial treatment may go for 10 or 12 years without symptoms, but when symptoms recur, further treatment may have little effect on the disease.

DR. FLETCHER AUSTIN: I have had occasion to see two or three cases whose presenting complaint was dysphagia. I wonder whether this is from mediastinal involvement?

DR. HOLINGER: Generally, yes. One of the characteristic initial sites of Hodgkin's is in the mediastinum. Compression of the esophagus by the large mediastinal mass causes dysphagia. This responds very rapidly to therapy, but the response may be only temporary.

DR. STANTON A. FRIEDBERG: I wish to thank the discussors. I would respond to Dr. Holinger's question by saying that I thought the nonulcerative characteristics of these lesions of the pharynx were just a curiosity. I do not believe that this is characteristic of such lesions, but in our group, despite huge masses, none of them were ulcerative. This observation has been made in the literature by others as well. I might say that of the 16 patients only 1 died as a result of local extension of the primary lesion.

Our experience with therapy has been identical with Dr. Holinger's. The lesions disappear rapidly after initial therapy, only to reappear in various parts of the body.

I should like to ask Dr. Wheelock if he has had experience in observing different types of lymphomas in the same person as he followed the patient over a long period of time?

DR. MARK C. WHEELOCK (by invitation): In a sense the leukemic infiltrate was merely a localized manifestation of the disease. The thought I meant to convey was that even at the time we made a diagnosis on some of these cases, we knew there were other regions involved. The point I wish to stress is the fact that the diagnosis in these instances was made at the time we had a piece of tissue from the region mentioned. In some cases the diagnosis had been made elsewhere by another pathologist.

Plasmacytoma is still maintained as a separate entity. On the other hand, we know it will often terminate as a plasma-cell leukemia. I think they behave a great deal like leukemia, and probably one day will be included in that group.

I might say that Dr. Friedberg's presentation brought out my own personal concept from the point of view of pathology—that is, not putting too much weight on the histologic aspects. I should like to say that in each of these cases, you are probably dealing with some form of lymphoma. It is well to suspect that particular entity in these lesions. In lesions of the pharynx, it is ridiculous to try to catalogue them as Hodgkin's, paragranuloma, granuloma, or sarcoma. I believe from the cases I have seen for microscopic study that ulcerations are not common. I have seen some ulcerations on the surface without a secondary inflammatory lesion. If there is no secondary inflammatory lesion, the diagnosis is difficult. My whole principle goes back to the fact that clinical aid in addition to histological substantiation is of great help in these cases.

I wish to say only one thing about therapy in these cases. I am not very impressed with chemotherapy. I think excision of the involved nodes with subsequent radiation therapy should be given a great deal of consideration. Although nitrogen mustard or steroid therapy may control the disease in the bone marrow, if hypoplastic anemia develops, the patient probably will have an ulcerative enterocolitis plus a mycotic infection in the lung, prostate, and/or kidney, which must be controlled. In such therapy, I should like to make a plea for a certain amount of consideration for the patient.

DR. WEISS: I wish to thank the essayists for their excellent presentations.

Abstracts from Current Literature

Ear

COMPLICATIONS OF MASTOIDITIS IN THE ANTIBIOTIC ERA. RUFUS C. MORROW, Ann. Otol. Rhin. & Laryng. 67:41-60 (March) 1958.

Relative to the present literature on mastoiditis and his own experience, the author points out that dangerous complications of mastoiditis are continuing to occur, often the penalty of reliance on antibiotics in the early stages of the disease.

Surgery has been classically indicated in the presence of postauricular swelling, mastoid tenderness, posterior canal wall sagging, or roentgen evidence of bony destruction. However, in view of present-day antibiotic therapy, surgery is indicated when acute mastoiditis subsides on antiobiotic therapy but there is persisting malaise, low fever, anemia, leukocytosis, pain in and about the ear, headache, or vertigo.

Nonsurgical management of meningitis with otitis media is justified if the meningitis and otitis are of simultaneous onset. However, if an ear infection responds to antibiotics but is followed by meningitis a week or so later, mastoid surgery then becomes indicated. Meningitis occurring early in antibiotic-resistant otitis media, late in the course of otitis media, or during chronic otitis media demands consideration of mastoid surgery.

The patient who suffers from recurring acute exacerbations of chronic otitis media of painless type but who develops, during an attack, pain and tenderness in the mastoid, headache, malaise, anorexia, low fever, vertigo, ataxia, or low leukocytosis should be looked upon with suspicion.

Of the 90 cases studied by Morrow, the causative organism was Proteus in three and Pseudomonas in one. This points up the ability of these common chronic ear infection organisms to produce dangerous complications at times.

STEELE, Kansas City, Kan.

THE EFFECT OF TRACTOR NOISE ON THE AUDITORY SENSITIVITY OF TRACTOR OPERATORS. DEAN M. LIERLE and SCOTT N. REGER, Ann. Otol. Rhin. & Laryng. 67:372-388 (June) 1958.

The authors studied 80 full-time tractor operators with negative ear histories, arriving at certain trends.

Tractor noise was found sufficiently high in intensity to produce high frequency (4,000 cps) losses in tractor operators with noise susceptibility if exposed over a period of several years. Attenuation of tractor noise can be greatly reduced by improved muffler design and utilization of better vibration control.

The 4,000 cps losses are more commonly found in the 30- to 60-year age group. The left ear is more frequently affected than the right ear. Since tinnitus is usual in the noise-susceptible person, it is advisable for those so affected to wear effective ear plugs while operating tractors.

STEELE, Kansas City, Kan.

THE PRESCHOOL NERVE-DEAF CHILD: STUDY OF ETIOLOGICAL FACTORS. BERNARD ZONDERMAN, Laryngoscope 69:54 (Jan.) 1959.

This is a very complete review of the literature on the subject of the nerve-deaf child together with the results of a study of 328 children examined by the author.

There are numerous causes. Heredity is one; consanguinous marriages among the deaf is another (recessive sporadic deafness). Prenatal causes include rubella, ototoxic drugs given the mother, infections, and γ-globulin given during pregnancy. At birth, injuries, anoxemia, and erythroblastosis fetalis are causative. After birth there are meningitis, measles, otitis media with labyrinthitis, mumps, and other diseases which may affect the hearing.

In all the series of cases studied, including Zonderman's, there is a large proportion, about one-third, of unknown etiology. One reason for this is the age of the child when first seen by an otologist. Hearing loss should be detected close to 6 months of age. If then

detected, the mother can begin speech training and lipreading at once, which is most important if the child is ever to acquire fluent speech.

HITSCHLER, Philadelphia.

PHENLYBUTAZONE AS AN ADJUNCT IN THE TREATMENT OF ACUTE OTITIS EXTERNA AND OTHER EDEMA OF THE EAR, NOSE, AND THROAT. MERRILL LINEBACK, Laryngoscope 69:96 (Jan.) 1959.

Traumatic or infectious edema of the ear, nose, or throat is rapidly reduced by the use of phenylbutazone (Butazolidin). With infection it is used with the usual antibiotics, etc., but without the use of enzymes or hormones. In acute primary external otitis, for example, the duration of the edema was reduced from six to nine to three to five days. However, one must be aware of possible allergy to it and the fact that it has a tendency toward producing peptic ulcer and anemia. (The reviewer has heard of many very severe toxic reactions from the use of phenybutazone).

HITSCHLER, Philadelphia.

Larynx

CARCINOMA OF THE LARYNX. GLENN E. SHELINE and ROBERT S. STONE, Ann. Otol. Rhino. & Laryng. 67:1066-1072 (Dec.) 1958.

The authors present a study of 124 patients with laryngeal carcinoma treated in a 10-year period. Ninety-six showed primary vocal cord lesions; twenty-six occurred supraglottically, and two, in the subglottic region.

Of the 96 cordal lesions, 58, or 60%, were controlled by radiation therapy. Two radiation failures were controlled by secondary laryngectomy. Thirty-one patients had cordal lesions which would have been suitable for cordectomy; instead, these were treated by radiation and twenty-four, or 80%, were alive and well at five years. Of the 43 cord lesions which would normally have required total laryngectomy, 67% were controlled by radiation alone. Of 19 patients with carcinoma too far advanced for control by laryngectomy, 5, or 26%, were salvaged by radiation therapy.

Most patients were treated through right and left lateral opposing portals, the dose being calculated in the midplane. The field size varied from 5 by 5 cm. for cordal lesions to somewhat larger sizes, depending upon the size and location of the lesion. Fifty-eight patients received a tissue dose of 5,500 to 7,000 r in 25-35 days. Forty-four patients received 5,000 to 6,000 r in 30-40 days. Eighteen patients were treated with 5,500 to 6,500 r in 35-40 days. In most, there was an attempt to deliver a midline dose of 5,000 to 6,000 r, although mucosal and skin reactions, as well as observation of the lesion, modified the planned dose in some instances during the course of therapy.

STEELE, Kansas City, Kan.

COMFORTABLE BRONCHOSCOPIES USING THE SITTING POSITION. J. K. POPPE, Ann. Otol. Rhin. & Laryng. 67:1202-1206 (Dec.) 1958.

Poppe advocates the sitting position for bronchoscopy, using topical anesthesia in all patients save infants, children, or those who are comatose. A valuable method in the orthopneic patient, others find it less traumatic and frightening, according to the author. Effort and time on the part of the hospital staff is saved, as the patient remains in a wheel chair throughout the procedure. Many of the author's patients were examined in outpatient facilities, eliminating a costly trip to the operating room and admission to a bed.

STEELE, Kansas City, Kan.

PRIMARY BRANCHIOGENIC CARCINOMA. N. P. COLLINS and M. T. EDGERTON, Cancer 12:235-239 (March-April) 1959.

As is well known, there have been some doubts raised regarding the existence of squamous-cell carcinoma occurring in the neck as a result of change in the lining of a branchial cleft cyst. The present authors report a case which they believe fulfills the requirements of such a diagnosis. In this particular instance, squamous-cell carcinoma was found in the wall of a deep-seated lateral cervical cyst that had a stratified squamous lining. The actual transition from uniform stratified squamous epithelium to squamous carcinoma is well illustrated by the histological preparations. All efforts were made, over a five-year period, to detect any primary neoplasm in the upper passages. It is particularly important in such situations to

rule out the possibility of thyroid carcinoma. Although the patient under consideration died from recurrent carcinoma approximately five years after the onset of symptoms, there was never any lesion in the upper respiratory or digestive tract that could possibly have been a primary tumor. The authors believe that careful study and extensive surgery might be responsible for the detection and cure of more lesions of this type.

FRIEDBERG, Chicago.

CANCER OF THE EPIGLOTTIS. H. J. SHAW and S. S. EPSTEIN, Cancer 12:246-256 (March-April) 1959.

The authors review the anatomy and the lymphatic supply of the epiglottis and call attention to the fact that a high percentage of patients show evidence of cervical lymph node involvement at the time the original diagnosis of cancer of the epiglottis is made. It is frequently difficult to assess the exact origin of a tumor of the epiglottis, but the authors point out the slow progress of those which arise near the tip and the infrequent likelihood of lymphatic metastases. Lower down on the laryngeal surface or further toward the ary-epiglottic folds, one notes the greater danger of rapid and extensive spread. The possibility of bilateral involvement of cervical lymphatics, either by metastatic spread or from an extension into the preepiglottic space, and of involvement of the tongue base, deep neck tissues, and strap muscles of either side are additional methods of spread. It is emphasized that there is virtually no mechanical barrier to the infiltration of the preepiglottic space by a tumor which involves the fixed part of the epiglottis.

Epiglottic cancers constituted almost 24% of the extrinsic laryngeal cancers in a large series studied and 11% of all laryngeal cancers. A mean total of 56% of the patients in the series had clinical involvement of cervical lymph nodes at the time of initial treatment.

Of all tumors examined, 80% were squamous carcinomas. The treatment used in this series consisted exclusively of irradiation and surgery was reserved, in general, for the treatment of irradiation failures. Since the best result recorded thus far in epiglottic cancer is a 17% five-year survival rate, and, since the authors of this article point to a 15% five-year survival, it is concluded that in the majority of cases nothing less than total laryngectomy, combined with bilateral neck dissection, is likely to offer a better chance of cure than is now available. In reference to survival, it was noted that all patients without clinical involvement of neck nodes on first examination have almost twice the survival rate of those having such involvement. The early improved results in cancer surgery elsewhere in the larynx is thought to offer additional hope that better chance of cure for epiglottic cancer is available.

FRIEDBERG, Chicago.

LARYNGOFISSURE IN THE TREATMENT OF LARYNGEAL CARCINOMA: A CRITICAL ANALYSIS OF SUCCESS AND FAILURE. MALCOLM H. MCGAVRAN, HARLAN J. SPJUT, and JOSEPH OGURA, Laryngoscope 69:44 (Jan.) 1959.

Seventy-nine consecutive cases with authenticated cancer of the larynx are studied. The criteria for success include (1) laryngofissure the only definitive therapeutic procedure, (2) five years without recurrence, (3) no evidence of cancer at necropsy if patient died in less (though not immediately postoperatively) or more than five years. Among failures are included those who died in less than five years without necropsy and those who were lost to follow-up. With this criteria the success rate was 57%.

The operation should include in a single block the true and false cords, the ventricle, the vocal process and a small area of the subglottis. Recently, the entire anterior commissure and a part of the opposite cord are included.

Functional results are fair to good. Hoarseness is always present. Results in those cases where the pathologist reported "tumor at the margin" are discussed.

After considering the functional result and the percentage of success it is concluded that irradiation therapy should supplement laryngofissure as the therapy of choice.

HITSCHLER, Philadelphia.

PATHOLOGIC CHANGES OCCURRING IN SEVERE REFLUX ESOPHAGITIS. R. N. MOERSCH and F. HENRY ELLIS JR. Surg. Gynec. & Obst. 108:476-484 (April) 1959.

After an excellent summary which deals with terminology and etiology of esophagitis, the authors summarize a series of cases in which resection of the esophagocardial region was performed. Thirty-six cases covering the years 1945 through 1956 were included in the

study. An attempt is made to classify the pathological changes as follows: Marginal esophageal ulceration was observed in a group of 23 patients, making up 64% of the entire series. This marginal esophageal ulceration was observed most commonly where hiatal herniation and incompetence of the cardiac sphincter were clinically evident. The ulceration in this group was superficial; only a short segment of the esophagus was involved, and there was concomitant shortening of the esophagus with stenosis and stricture.

The second type of pathology is referred to as extended linear esophagitis. This occurred in 9 cases, or 25% of the series, and the lesions were characterized by superficial inflammation which extended for several centimeters proximal to the esophagogastric junction.

Solitary marginal ulcer was observed in two patients, and in both of the specimens studied there were deep penetrating ulcers. It was impossible to designate this ulceration as either esophageal or gastric.

In a single case there was a deep ulceration in a columnar epithelial-lined esophagus, the so-called Barrett type of ulcer.

It was observed that inflammation in the esophagus is generally superficial even in the presence of severe stricture. The prognosis seems to be somewhat poorer in patients who have extension of the inflammatory process into the muscular sheath, although therapeutic results were not analyzed in the present study. The single instance of Barrett ulcer occurrence was considerably lower than might have been expected in view of the frequent reference to "ectopic gastric mucosa" in the esophagus.

FRIEDBERG, Chicago.

Nose

STRESS FACTORS IN RHINOLOGY. EDGAR E. POOS, Ann. Otol. Rhin. & Laryng. 67:1073-1077 (Dec.) 1958.

Poos emphasizes the importance of stress in rhinology, due to its effect on the autonomic nervous system, endocrine system, neurovascular system, capillary permeability, electrolytes, acid-base equilibrium, and water balance.

STEELE, Kansas City, Kan.

LETHAL MIDLINE GRANULOMA: IS IT A PATHOLOGICAL ENTITY? HERSCHEL H. BURTON, Laryngoscope 69:1 (Jan.) 1959.

The gradual destruction of the nose, palate, and face has been a challenge to medicine since first being recognized, in 1896. It begins with nasal obstruction and watery discharge, progresses to a slow but continuous necrotic stage lasting some 10 to 18 months, and terminates with rapid advancing necrosis and exhaustion of the patient. In the second stage, the patient feels comparatively well. It is in this stage that active therapy should be undertaken.

Differential diagnosis is sometimes difficult. It is discussed in detail. Pathogenesis seems to relate to immunological or stress effect on tissue changes. It has been demonstrated that if a tissue has been sensitized and antigen injected, a granuloma will form.

There is no uniform pathologic picture. As progress continues, more and more cases of so-called lethal midline granuloma are being diagnosed pathologically as specific diseases.

Sometimes cortisone reduces the inflammatory reaction sufficiently to enable the pathologist to recognize the nature of the underlying tumor.

Supportive measures, including blood transfusions, designed to raise the defensive mechanisms, together with alertness as to causative factors, offer the best means of combating these lesions.

HITSCHLER, Philadelphia.

Pharynx

NICOTINE STOMATITIS OF THE PALATE. WILLIAM H. SAUNDERS, Ann. Otol. Rhin. & Laryng. 67:618-627 (Sept.) 1958.

The clinical appearance of nicotine stomatitis may take the form of redness of the orifices of the mucus glands of the palate or may be characterized by papular changes. Occasionally, ulcerative or granulomatous lesions are produced by nicotine irritation. A fine, pink, wart-like or papillary formation over much of the soft palate mucosa is commonly found.

Since this disorder is virtually asymptomatic, the mucosal changes caused by the disease are usually overlooked unless the ulcerative or granulomatous types are present.

Microscopic study of nicotine stomatitis in the early stages shows increase in keratin which produces the white or gray mucosal coloring. Later, when papule formations occur, there is dilatation of the ducts of the mucus glands and tissue infiltration with lymphocytes is found. Metaplasia of the duct lining to squamous epithelium may occur.

Treatment is abstinence from smoking.

STEELE, Kansas City, Kan.

ORAL AND FACIAL CANCER. HAROLD H. SAGE, New York J. Med. 59:1113 (March 15) 1959.

This is another in the series of cancer alerts broadcast over WNYC under the aegis of the New York Academy of Medicine and the New York City Cancer Committee.

Cancers of the mouth and face are usually epidermoid in type. Since these are so "getatable," early diagnosis offers a chance of early cure by surgery or radiation or both. The surgeon aims to remove the diseased area en bloc. Facial cancer includes the skin, sinuses, nasopharynx, and parotid and submaxillary glands. Oral cancer is in the lips, buccal mucosa, gums, palate, floor of the mouth, and tongue. Dividing the tongue into anterior, lateral, and base areas calls for special study in each instance as to diagnosis and treatment. If metastasis has not occurred the five-year cure is in prospect. Variations in sensitivity and rapidity of growth are wide. The dentist has a great opportunity to see incipient cancer in the mouth and should always be on the lookout for malignancy. The public which is so fond of sun tan needs to know that cancer may develop after overexposure. Cancer in the paranasal sinuses is often insidious and unexpected before surgery. Cancer in the parotid is difficult to remove by surgery, since the dissection must be wide and it involves very important structures, e. g., the carotids. X-ray and radium may be effective for postoperative fields and, of course, for advanced cancers. It requires special knowledge and technique.

VOORHEES, New York.

Miscellaneous

BRONCHIAL ADENOMA IN CHILDHOOD. GORDON F. VAWTER and CHARLES F. FERGUSON, Ann. Otol. Rhin. & Laryng. 67:1113-1125 (Dec.) 1958.

The triad of hemoptysis, recurrent pneumonitis and chronic cough, and bronchial occlusion should suggest bronchial adenoma in children. This is a rare lesion in the 0-16 age group. The authors were able to find but 20 cases reported in the literature. Bronchoscopy proves the existence of the lesion, and bronchography will detect the presence of bronchiectasis.

Treatment has been by endoscopic removal or coagulation, bronchotomy, lobectomy, or pneumonectomy and radiation therapy. The single case reported by the authors was successfully treated by lobectomy.

STEELE, Kansas City, Kan.

STUDIES IN SIALOLITHIASIS: THE STRUCTURE AND MINERALOGICAL COMPOSITION OF SALIVARY GLAND CALCULI. IRVING BLATT, REYNOLDS DENNING, JAMES H. ZUMBERGE, and JAMES H. MAXWELL, Ann. Otol. Rhin. & Laryng. 67:595-617 (Sept.) 1958.

Adopting the analytical methods of the geologist, mineralogist, and the microanalytical chemist, Blatt and co-authors studied 30 salivary calculi. They were found to show a laminated structure in which concentric shells of calcareous mineral, apatite, alternate with layers of organic resinous material, which also forms the outer covering of the stone. The central core, or nucleus, is a spheroid mass of apatite, without evidence of foreign body or organism in the calculi examined.

The laminated feature of the stone indicates a growth pattern in which physicochemical relationships alternate between periods of calcareous deposit and accumulation of organic, resinous material. There is no apparent cause for the initial stage of accretion.

Apatite, the crystalline component of the salivary gland calculus, makes up a group of biologically occurring complex phosphate compounds. Carbonate-apatite appears to be the apatite in the salivary calculus. A small amount of calcite is also present.

STEELE, Kansas City, Kan.

BUCCAL MUCOSAL TESTS IN PATIENTS WITH CANKER SORES (APHTHOUS STOMATITIS). LOUIS TUFT and LEONARD S. GIRSH, J. Allergy 26:502 (Nov.-Dec.) 1958.

The etiology of canker sores (aphthous stomatitis) is not known. An allergic etiology has been considered but has not been proved. A pilot study was carried out in which four organic acids commonly found in foods were applied directly to the anterior buccal mucosa. The organic acids tested were citric acid, tartaric acid, acetic acid, and lactic acid. Citric and tartaric acid were tested in powder form. Acetic acid was tested as a 5% solution, and lactic acid was tested as O.1 N solution.

Tartaric acid and lactic acid gave negative tests and were not considered of etiological importance. Canker sores were produced in patients with a positive history of aphthous stomatitis with citric and acetic acids. A history of canker sores was more frequent in allergic patients than in nonallergic patients.

This study suggests a possible allergic basis for the occurrence of canker sores in some patients.

MISSAL, Cleveland.

FOOD SENSITIVITY. STANLEY S. FREEDMAN and WILLIAM SELLARS, J. Allergy 30: (Jan.-Feb.) 1959.

A study was made of 150 consecutive cases of "allergic" children to determine the incidence of food sensitivity and to assess the role that food sensitivity plays in various allergic disease entities. Ninety-seven of the group had atopic eczema and nonseasonal asthma, and of this number 47 had proved food sensitivities. The manifestations of their food sensitivities were unrelated to the asthma or eczema in most instances. The majority of the foods showed a low incidence of correlation between proved clinical food sensitivity and positive skin tests. In almost every case the food allergy was suggested by the history and not by skin tests. The authors observed that the use of raw food for skin testing gave stronger reactions than their corresponding commercial extracts.

MISSAL, Cleveland

EUSTACHIAN TUBE FOREIGN BODY: REPORT OF A CASE. W. E. COMPERE JR., Laryngoscope 69:90 (Jan.) 1959.

Slag in the external canal is often associated with tympanic perforation. It is therefore unwise to irrigate the canal to attempt removal because of the danger of washing the slag into the middle ear cavity and even into the Eustachian tube. This presumably happened in the case reported. Otitis media and pain ensued. The slag was dislodged by a Eustachian bougie and then blown into the middle ear cavity, where it was extracted with a suction tip.

HITSCHLER, Philadelphia.

ANESTHETIC MANAGEMENT OF CONGENITAL ATRESIA OF THE ESOPHAGUS AND TRACHEOESOPHAGEAL FISTULA. RITA GOLDMAN JACOBS and E. M. PAPPER, New York J. Med. 59:995 (March 15) 1959.

Both authors work in the Presbyterian Hospital-College of Physicians and Surgeons, Columbia University. The paper was read before the 152d Annual Meeting of the Medical Society, State of New York, May 16, 1958.

The report concerns 72 patients operated upon for atresia or fistula between 1950 and 1956 at Babies Hospital. Of these, 41 lived (57%). Many are premature babies (here, 45%) and often have additional anomalies. Full-term infants were 31 in number; survival was 81%. Anesthesia is almost the chief consideration with reference to use of the endotracheal tube. Early diagnosis and operation are highly important. At birth it would be well to pass a catheter into the esophagus to make sure of patency. Drawbacks to survival are prematurity, pulmonary infection, malnutrition, dehydration, and other congenital defects, all of which must be intensively treated preoperatively and postoperatively.

ABSTRACTER'S COMMENT: This is "heroic surgery" at its best. Surgery *must* be done or the patient will die, and so one has much to gain. To succeed with such a high rate of survival must be a source of great satisfaction. But this is a specialty within a specialty. It is no field for a novice.

VOORHEES, New York.

SUPPURATION MASKED BY ANTIBIOTICS. JACOB J. ROBBINS, New York J. Med. 59:1121 (March 15) 1959.

Surgery is the only method of choice in the presence of suppuration. An unwise use of antibiotics may mask the condition and lead to dangerous delays. Three cases are reported here, but the third is of especial interest to the ENT man. A 20-year-old U. S. A. F. enlisted man had "stuffiness and rhinorrhea" three weeks before admission to the Navy Hospital at St. Albans, N. Y. Nose drops and cleansing plus chlortetracycline were administered, but nausea, vomiting, and intermittent fever persisted. The work-up was very thorough, but showed little save the blood picture, with 16,000 white cells and 80% polymorphonuclear cells. There were papilledema, bradycardia, and lethargy. The electroencephalogram was only suggestive of brain abscess. However, both frontals were cloudy and sensitive to pressure. Surgery revealed a right frontal subdural abscess from which 85 cc. of greenish-yellow pus was evacuated. Penicillin, chloramphenicol, polymyxin, and streptomycin had been continued. Eventually the hemolytic Streptococcus was isolated. The patient made "a complete recovery."

ABSTRACTER'S COMMENT: This seems to have been a sinus infection from the beginning and was probably recognized as such, but only of a moderate degree. The antibiotics gave relief and masked the subtle suppuration which was going on in the sinuses. This also has happened in patients with mastoiditis. The danger of being "fooled" has been reported in the literature by many surgeons. It needs to be reemphasized over and over.

VOORHEES, New York.

Books

BOOK RECEIVED

Treatment of Cancer and Allied Diseases. Volume 3: Tumors of the Head and Neck. Second Edition. Edited by George T. Pack, M.D., and Irving M. Ariel, M.D. Price, $30. Pp. 781, with 1,028 illustrations. Paul B. Hoeber, Inc. (medical book department of Harper & Brothers), 49 E. 33d St., New York 16, 1959.

Headache: Diagnosis and Treatment. Edited by Arnold P. Friedman, M.D., and H. Houston Merritt, M.D. Price, $8. Pp. 411, with 16 illustrations, plus tables and sample charts. F. A. Davis Co., 1914 Cherry St., Philadelphia 3, 1959.

BOOK REVIEWS

Hearing: A Handbook for Laymen. By Norton Canfield, M.D. Price, $3.50. Pp. 214. Doubleday & Company, Inc., Garden City, New York.

This book is written specifically and exclusively for lay people by an author who has had a vast experience in clinical otology and particularly rehabilitation. It provides a general perspective of all aspects of hearing and deafness. Attesting to its simplicity is the absence of any figures, and not a single audiogram appears in the book. It is specifically designed to help people who have hearing problems. Special emphasis is placed upon deafness in children and rehabilitation as well as hearing aids. Because there is no other book known to the reviewer that reaches the lay public on the same level as this one, he believes it has a great deal of usefulness and should be very well received by the general public.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. G. Arnold Henry, 170 St. George St., Toronto.
Secretary: Dr. Donald M. MacRae, 324 Spring Garden Rd., Halifax, N. S.
Meeting: Sheraton-Brock Hotel, Niagara Falls, Ontario, Oct. 9-10, 1959.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Association of Allergology

President: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago 11.
Secretary-General: Dr. José Quintero Fossas, Paseo 313, Vedado, Havana, Cuba

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. John R. Lindsay, 950 E. 58th St., Chicago.
Secretary-Treasurer: Dr. Louis Savitt, 3403 W. Lawrence Ave., Chicago 25.
Meeting: 24th Annual Congress, Palmer House, Chicago, Sept. 14-18, 1959.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Mexican Society of Otolaryngology

President: Dr. Rafael Giorgana
Secretary: Dr. Carlos Valenzuela, Monterrey 47-201, Mexico 7, D. F.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Paul H. Holinger, Chicago.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Victor R. Alfaro, Washington, D. C.
Secretary: Dr. Walter E. Heck, San Francisco.

American Academy of Ophthalmology and Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Verling K. Hart, 106 W. 7th St., Charlotte, N. C.
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3.
Meeting: Deauville Hotel, Miami Beach, Fla., March 15-16, 1960.

American Laryngological Association

President: Dr. William J. McNally, Montreal.
Secretary: Dr. Lyman G. Richards, Wellesley Hills, Mass.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Meeting: Deauville Hotel, Miami Beach, Fla., March 18-19, 1960.

SECTIONS:

Eastern.—Chairman: Dr. Joseph L. Goldman, New York.
Southern.—Chairman: Dr. James T. King, Atlanta, Ga.
Middle.—Chairman: Dr. Alfred J. Cone, St. Louis, Mo.
Western.—Chairman: Dr. Robert C. McNaught, San Francisco, Calif.

American Rhinologic Society

President: Dr. Kenneth H. Hinderer, 402 Medical Arts Building, Pittsburgh 13.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Oct. 8-9, 1959, Illinois Masonic Hospital, Chicago.
Annual Meeting: Oct. 10, 1959, Belmont Hotel, Chicago.

American Otological Society, Inc.

President: Dr. Robert C. Martin, 384 Post St., San Francisco 8.
Vice-President: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Meeting: March 13-14, 1960.

American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Raymond S. Rosedale, 610 N. Market Ave., Canton 2, Ohio.
Secretary-Treasurer: Dr. Joseph G. Gilbert, 75 Barberry Lane, Roslyn Heights, N. Y.
Meeting: Oct. 11, 1959, Conrad Hilton Hotel, Chicago, and March 6-13, 1960, Deauville Hotel, Miami Beach, Fla.

American Society of Facial Plastic Surgery

President: Dr. Oscar J. Becker, Chicago.
Vice-President: Dr. Sam H. Sanders, Memphis.
Secretary: Dr. Samuel M. Bloom, 123 E. 83d St., New York 28.
Treasurer: Dr. Joseph C. Miceli, Brooklyn.
Meeting: Oct. 15, 1959, Chicago.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Joseph W. Hampsey, Grant Building, Pittsburgh 19.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6.

Otosclerosis Study Group

President: Dr. E. P. Fowler Jr., 180 Fort Washington Ave., New York 32.
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Society of Military Otolaryngologists

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1. Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics, ed. 2, New York, The Macmillan Company, 1955, p. 69.

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*(Binocular head supplied, with 12.5X lens, for an additional $275.00).

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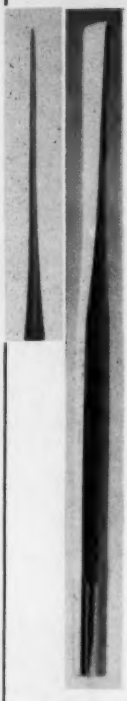

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Showing full Chisel in ¾ view and left upper illustration in side view.

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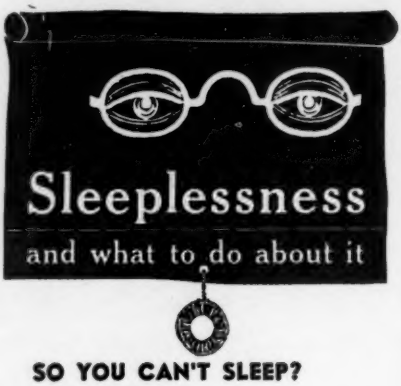

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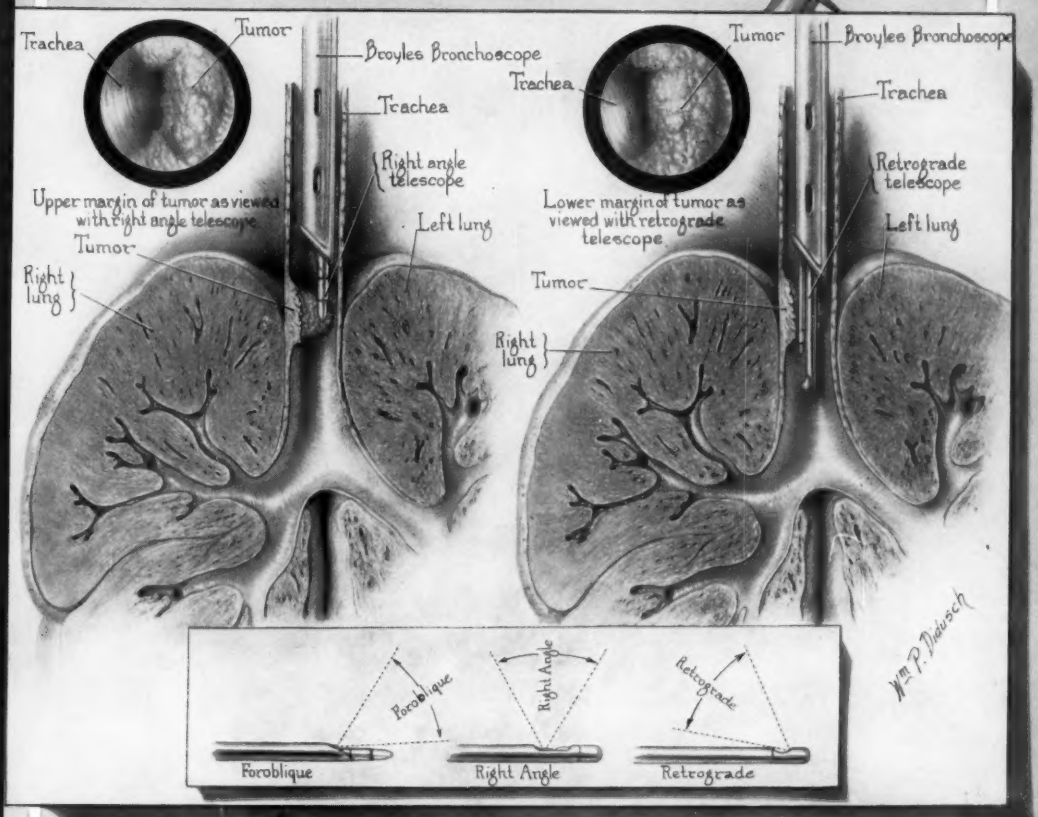

The illustration above shows the Broyles Bronchoscope and the use of right angle and retrograde telescopes in evaluating a tumor of the trachea.

The Broyles Bronchoscope with the right angle, the foroblique, and the retrograde telescopes provides accuracy in the proper evaluation and subsequent treatment of lesions of the trachea.

Views heretofore never obtained are now possible with the combination of telescopes. Tumors can be outlined and the margins clearly defined, showing the degree of lateral spread and the characteristics of infiltration so vital in establishing a comprehensive study.

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